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Compendium of Vegetable Crops Diseases



INDIAN PHYTOPATHOLOGICAL SOCIETY

Title: Compendium of Vegetable Crops Diseases

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Preface

Diseases are most limiting factor in vegetable production. There has been a demand from progressive vegetable growers, plant protection officials, agriculture advisors, private seed companies, subject matter specialist and research scholars since long for a comprehensive colour compendium on vegetable diseases. In absence of such compendium, identification is still difficult by the personnel engaged in different activities of vegetable production. Effective management packages cannot be advocated in absence of detailed symptomatology and pathological information. No compendium on diseases of vegetable crops is available so far with complete knowledge and information in India. Keeping this in view a colour technical bulletin entitled compendium of Vegetable diseases has been prepared. Different important diseases of vegetables caused by fungi, bacteria, phytoplasma, viruses and nematodes have been covered with information on common name of disease, pathogen name, symptoms, epidemiology and control measures. It has been prepared to assist clear diagnosis of the diseases and planning of integrated management strategy for sustainability of vegetable production. Considering the necessity of environmental safety, pesticide residue problem and cost-effective protection of the crop, it is desired to develop integrated disease management schedule for the whole year. The identification and management strategies advocated in this bulletin will serve the present and future need of vegetable producer for long-term efficacy and sustainability. We hope that this technical bulletin will serve to all needy personnel directly or indirectly related to vegetable production.

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Introduction

Vegetables are an important component of the human diet and their consumption are increasing continuously due to rich source of vitamins, minerals, crude fiber and shift to vegetarian food habit. Vegetable crops are more susceptible to diseases as compared to other crops. Various biotic factors like fungi, bacteria, viruses, phytoplasmas and nematodes are responsible for causing diseases in vegetables that result severe losses in yield and quality. In recent past, introduction of hybrids of vegetables created new disease problems with increased disease intensity. Disease pressure in the standing crop and spoilage caused by fungi and bacteria during transit, storage and marketing are the main business constraints of vegetable produce. Approximately 65 million tones pre-harvest losses in vegetables due to disease alone in the world has been estimated that accounts about 10.5 percent of the total produce of vegetables. Pre-harvest loss means, the produce either never produced because of indirect interference of pathogen in host physiology or directly destroyed by disease before harvesting. Post-harvest losses are equally important because most of the vegetables are highly perishable in nature and easily colonized by the microorganisms. The fungicides in addition to non-target effects are becoming expensive year after year and losing their effectiveness due to development of resistance in pathogen strains. Most of the new generation pesticides are systemic in nature which leads to certain level of toxicity in the plant system and affecting health hazards. Further, it disturbs complete ecology of the microbial diversity of whole ecosystem.

Disease scenario in vegetable crops has changed due to extensive use of exotic hybrids, intensive and monoculture of vegetables. Disease pressure of soil-borne pathogens like *Pythium*, *Sclerotinia*, *Rhizoctonia*, *Fusarium* and *Sclerotium* are increasing and engulfing almost all vegetable crops causing very severe crop loss every year. Virus associated diseases caused by cucumber mosaic virus, tospovirus, begomovirus, potyvirus and illarvirus in vegetables caused huge losses every year to different vegetable crops. Recently, phytoplasma diseases are also emerging as very important diseases in many vegetable crops and causing significant economic losses. Soil borne bacterial pathogen, *Ralstonia* causes wilt disease in solanaceous vegetables is responsible to shift of cropping pattern in a locality. The off-season vegetable growing practices have prolonged the survival period of pathogens in the field. The problem of seed-borne diseases like black rot of cole crops, bacterial blight of tomato and cucumber mosaic virus in cucurbits and their inoculum has increased throughout the country.

Open General License (OGL) and General Agreement on Tariff and Trade (GATT) agreement results in an easy import policy of vegetable seeds from other countries. Free movement of susceptible seeds and planting materials by private seed companies, scientists and farmers has been observed throughout the country without proper domestic quarantine regulations. Multinational vegetable seed companies have been easily importing the seeds and susceptible planting materials from different countries, which further increased disease pressure and their inoculum in vegetables. All these factors have led to new dimension in research for diseases in vegetable crops. In recent years the research, demonstration and practical use of integrated disease management has been taken in real application in agriculture particularly for vegetable production.

Integrated Disease Management: Basic Concepts

In the recent past, the term management has replaced control. Control means absolute check of a disease, which is neither true nor possible. No pathogen is completely eradicated from a natural ecosystem but only its population can be minimized up to economic injury level. Disease management does not employ against the only pathogen but also considers the other two components of disease triangle i.e. host and the environment that gives the whole concept of integrated disease management (IDM). The main objective of IDM is to manage the losses below an economic injury level and minimize the recurrence of a disease by interrupting in the disease cycle. Integrated management practices should be planned in such a way in the normal cultural practices that may be effective against more than one disease. Integrated disease management lies in pathogen, host, time, and environment. Pathogen management involves reduction in inoculum, eradication, and prevention of inoculum while management of host involves improving plant vigor, inducing resistance through nutrition and genetic manipulation and protection by chemical means. The time factor could be managed by adjustment of sowing/planting date so that favorable time of disease should not coincide without any adverse effect on yield. Management of environments involves water, soil and crop management. The principles of IDM are based on avoidance, exclusion, eradication, protection, and host resistance. Avoiding disease by planting at times when or in areas where inoculum is ineffective due to environmental conditions. It could be also achieved by selecting geographic area for disease free seed production, using disease escaping varieties, selection of disease-free seeds and modification of cultural practices. Sowing of okra up to first week of July is effective in managing *Cercospora* leaf blight of okra. Summer crop of cucumber is almost free from downy mildew. Exclusion means preventing the inoculum from entering or establishing in the field or area where it does not exit. Seed certification, seed treatment, plant quarantine and eradication of insect vectors are effective methods to check disease spread. Eradication is applied when pathogen has already been entered in area or crop despite the above-mentioned precautions. At this condition, biological control methods, crop rotation, rouging, eradication of alternate and collateral hosts, sanitation, soil treatments, heat and chemical treatments of diseased plants can be applied. Crop rotation with cereals and fallowing is quite effective to reduce incidence of root knot nematode, wilt of pea, *S. rolfsii* and *Verticillium dahliae*. Composted municipal sludge when incorporated in the soil at high rate (10%w/w) reduces *Aphanomyces* root rot of pea, *Rhizoctonia* root rot of bean and radish, *Fusarium* wilt of cucumber and *Phytophthora* crown rot of pepper. Protecting infection by creating a chemical toxic barrier between the plant surface and the pathogen is very much applicable for checking secondary inoculum and infection for those diseases, which have very high infection rate. It includes chemical spray on the diseased plants and for insect vectors, use of barrier and trap crops. Host resistance is the best method of disease management where moderately resistant and tolerant lines can be developed with great success without reducing qualitative and quantitative yield attributes.

Open General License (OGL) and General Agreement on Tariff and Trade (GATT) agreement results in an easy import policy of vegetable seeds from other countries. Free movement of susceptible seeds and planting materials by private seed companies, scientists and farmers has been observed throughout the country without proper domestic quarantine regulations. Multinational vegetable seed companies have been easily importing the seeds and susceptible planting materials from different countries, which further increased disease pressure and their inoculum in vegetables. All these factors have led to new dimension in research for diseases in vegetable crops. In recent years the research, demonstration and practical use of integrated disease management has been taken in real application in agriculture particularly for vegetable production.

Integrated Disease Management: Key Strategies

Success of plant disease management approach is only possible when we consider the following principles and precautions:

- ❑ Avoid indiscriminate use of fungicides and antibiotics and do not mix incompatible pesticides, phytohormones and micronutrients at same time.
- ❑ Soil is a reservoir of harmful as well beneficial microorganisms therefore, soil health should be properly managed by timely tillage, summer ploughing, green manuring, optimum C:N ratio, balanced macro and micronutrients, aeration etc.
- ❑ Seed health must be maintained up to prescribed standard and ensure that seed should be free from internal and external pathogen, contamination of any infected crop debris, sclerotial bodies etc.
- ❑ Field sanitation includes removal and burning of infected crop debris, alternate and collateral weed hosts etc.
- ❑ Crop rotation is very important for the soil borne diseases where non-host crop should be selected for a pathogen.
- ❑ Avoid off-season vegetable and its intensive cropping because it prolongs the perpetuation period of a pathogen and do not break the life cycle of the pathogen.
- ❑ Knowledge of correct diagnosis of the disease and the disease cycle of the pathogen is very important to apply different disease control methods at critical phase of life cycle for maximum efficacy.
- ❑ Selection of effective and broad-spectrum fungicides, optimum dose of the fungicide, site of application, mode of spray and droplet size affects the efficacy of fungicides.
- ❑ Application of biocontrol agent particularly native antagonist along with FYM or any organic matter should be maximized. At the same time insensitive and compatible fungicides to these antagonists must be applied.
- ❑ Always prefer to grow tolerant varieties and hybrids. However, it is difficult to get resistance against the diseases with all desirable traits in hybrid.
- ❑ Proper post-harvest management practices should be adopted to avoid any rotting of vegetable and seed infection.

Tomato

Introduction

Tomato (*Solanum lycopersicum*) is an important horticultural crop belonging to the family *Solanaceae*, cultivated for its edible fruit across the globe. The word “tomato” is derived from the Nahuatl word *tomatl*. The probable centre of origin of wild tomato is South America and it was domesticated in pre-Columbian Mexico. Tomato crop ranks second in the world vegetable production after potato and ranks third in India after potato and onion. The major tomato growing nations include China, India, Italy, USA, Turkey, and Egypt. The total worldwide production of tomato is 163.96 million tonnes per annum (FAO, 2016). India ranks second in total production after China. In India, the major tomato growing states include Maharashtra, Bihar, Uttar Pradesh, Karnataka, Orissa, Madhya Pradesh, Andhra Pradesh and Assam.

Tomato is one of the widely cultivated vegetable of India. It is an important vegetable crop regarding nutrition and income. Tomatoes fruits are utilized as fresh fruits, eaten raw in salads, or as cooked vegetables. A large proportion of tomato crop is utilized as processed products such as canned tomatoes, ketchup, soups, sauces, tomato juice, puree, chutneys, paste, pickles, dehydrated pulp etc. Tomatoes are nutritionally rich as they are a good source of vitamin C, A, B complex, potassium, minerals like calcium, sodium and organic acids viz., acetic, formic and citric acids. The attractive red colour of the fruit is due to presence of pigment viz. lycopene. Tomato is considered as good appetizer and acts as good remedy for prevention of constipation.

Tomato is a warm-season crop but can be grown throughout the year under greenhouse conditions. It grows well at an average temperature of 21 to 23°C. Heavy rainfall and prolonged dry spell exhibits detrimental effect on growth and fruit development. The overall production and productivity of tomato crop is hindered by a number of diseases, insect-pests and physiological disorders. Among all these, fungal diseases are a major limiting factor in tomato production. The major diseases affecting tomato production are damping off, collar rot, late blight and early blight.

Name of disease: DAMPING OFF

Geographical origin, distribution and crop losses: The term ‘Damping-off’ is coined during the early nineteenth century, and it represents one of the oldest nursery problems worldwide and the term is generally used for the death of seedlings, either before or after emergence from the soil. It is one of the most serious problems faced by the farmers in raising tomato seedlings in nursery beds. Damping off may affect 5 to 80 per cent of the tomato seedlings, thereby causing huge economic loss to the farmers (Lamichhane et al. 2017). Many farmers depend on producing early tomatoes for the market to capture profit but due to the disease there is a set-back of one or two weeks which usually does not reduce the yield, but it greatly reduces the net returns, because of the low price of late tomatoes. The additional cost of replanting also results in huge economic loss.

Symptoms: There are two types of damping-off, one is pre-emergence and other is post-emergence damping-off.

Pre-emergence damping-off: In case of pre-emergence damping-off, seeds rot or seedlings decay occur just before their emergence from the soil. In general, random patches of poor seedling emergence are the indication of pre-emergence damping off (Fig. 1).



Fig 1. Pre-emergence damping off

Post-emergence damping-off: In case of post- emergence damping-off, seedlings decay, wilt, topple down on the nursery bed occurs and the seedlings ultimately die just after emergence. The hypocotyl of the seedling becomes water soaked, discolored at the surface of the soil, stems become thin, turns gray, brown or black after emergence. If the plants survived, the hypocotyl and roots remain infected and the surviving plants show stunted growth, and affected areas often show uneven patches of plant growth.

Causal organism

Damping-off is caused by several soil-borne fungi including *Pythium*, *Phytophthora*, *Rhizoctonia* and *Fusarium*.

Key Characteristics: *Pythium* is a parasitic oomycete characterized by coenocytic hyphae without septations. *Phytophthora* produces coenocytic, multinucleate mycelium and the sporangia are hyaline, lemon shaped with a distinct papilla at the distal end and borne singly at the tip of alternately branched sporangiophores. *Rhizoctonia* belongs to the class Deuteromycetes which can survive as mycelium, sclerotia or basidiospores. Fungus is characterized by the structure of hyphae with a constriction at the point of branching and right angles of the hyphae. *Fusarium* produces extensive cotton like mycelium in culture, conidiophores variable, slender, branched irregularly or bearing a whorl of phialids, single or grouped into sporodochia; conidia hyaline, principally of two kinds, macroconidia are several celled, slightly curved at the pointed ends; microconidia 1 – celled, ovoid or oblong, borne singly or in chains.

Disease cycle and Favourable conditions

The pathogens survived or spread primarily by soil, seed and water and secondarily by conidia through rain or wind. The infection is most common in cool conditions, but *Phytophthora* and *Rhizoctonia* can infect seedlings in warmer soils also. As and when tomato seedlings reach the 2 to 3 leaf stage, they are no longer susceptible to the infection by *Pythium* or *Rhizoctonia* but *Phytophthora* can infect tomato plants at any stage of the growth. The disease is favoured by high humidity, poor soil drainage resulting in wetness of soil, low soil temperature, cloudiness and low temperatures below 24° C continuously for few days. Crowded seedlings also hamper plant growth and increase the disease.

Management

Damping-off management requires an integrated disease management (IDM) approach combining both preventive and curative management approaches.

Avoidance: Damping-off can be avoided by sowing seeds in well-drained, well-prepared raised seed bed; avoiding overcrowding, excessive shade, over watering, or over fertilization or by transplanting disease free seedlings. Avoid excessive application of farm yard manure and nitrogen fertilizer which makes seedlings succulent.

Biological Management: Seed treatment with extracts of *Lantana camara*, neem cake and cow urine can be effectively used for the management of damping-off of tomato in organic farming. FYM @5 q/ha + seed

treatment with *Trichoderma harzianum* @6gm/kg seed + seed treatment with *Pseudomonas fluorescens* @6 gm/kg seed also provide an effective management of the disease.

Chemical treatment: Soil drenching with Copper oxychloride @0.2% or Bordeaux mixture @1% can be effective. Metalaxyl @0.2% can also be sprayed for management of the disease as the last option since chemical treatment leads to the high risk of environmental pollution.

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Name of disease: EARLY BLIGHT

Geographical origin, distribution and crop losses: Early blight is one of the most common disease of tomato distributed throughout the world. It is considered as weed of tomato field due to its wide adaptability under various climatic conditions. However, it is more destructive in tropical and subtropical regions. It was reported that 1% increase in the intensity of the disease can cause decline in yield by 1.36% and complete failure of crop may occur under severe conditions (Pandey *et al.*, 2003).

Symptoms: Tomato is affected by two species of *Alternaria*. Early blight of tomato is caused by *Alternaria solani* with 40-45% average incidence. This is most destructive disease and prevalent all over the country. The yield loss was recorded 80-86% in experimental field due to early blight alone. *A. alternata* f.sp. *lycopersici* is another species causing leaf blight with 10-15% average incidence every year. Irregular leaf spots mostly appear from marginal portion of leaves. These lesions are having prominent yellowing on their outer margin due to host specific AAL toxin produced by the pathogen. Mostly disease appears in vegetative phase of plant growth before flowering. Symptoms of early blight appear on all above ground parts of the plant. Leaf spot symptoms are scattered, brown with concentric rings like a target board and often with chlorotic halo (**Fig. 2**). In humid weather, the spots coalesced and enlarged. Girdling of the stem at the base of the seedlings and plant can be seen. The stem lesions are usually restricted to one side of a stem and become elongated and



Fig.2 Early blight of tomato



Fig.3 Symptoms of early blight on tomato fruits

sunken. Primary infection occurs on blossoms. Fruit symptoms initiate from calyx and pedicels. Gradually, it progresses on apical portion of fruit (stem-end) as a spot and radiate out from the attachment between calyx and fruit. Fruit spots are dark brown, depressed firm and with distinct concentric rings (Fig.3). The pathogen is soil borne as well as seed borne in nature.

Causal organism: *Alternaria solani*

Key characteristics: The pathogen produces septate and darkly pigmented mycelium which darkens with age. The conidia are borne on distinct conidiophores singly or in a chain of two. The conidia are usually long beaked bearing 9-11 transverse septae. The isolates of the pathogen produces diffusible yellowish to reddish pigments on potato dextrose agar media.

Disease cycle and favourable conditions: The pathogen primarily overwinters on infected crop debris. Thick walled resistant spore i.e., chlamydospores have also been reported but occur infrequently. Under mild climatic conditions, the pathogen thrives on volunteer potato, tomato plants as well as other wild solanaceous plants viz., nightshade and horsenettle. Secondary spread of the disease occurs from airborne conidia dispersed mainly by wind and occasionally by rain splash and overhead irrigation.

The optimum temperature of 24-29°C and high relative humidity are conducive for the infection. The conidia germinates at an optimum temperature of 28-30°C in presence of free moisture. Desiccated germ tube possess ability to renew their growth upon rewetting. Thus, infection may occur under alternating dry and wet conditions and the pathogen penetrates the leaf either directly or through stomata. The incubation period of the pathogen depends on several factors such as leaf age, susceptibility of cultivars and environmental conditions. Early blight disease occurs primarily on aging plant parts. Sporulation requires a long wet period but can also be induced under alternating dry and wet conditions. Conidiophores emerge during wet night followed by day light and dry conditions induce spore formation, which are produced on second wet night.

Management

Resistant cultivars: A number of varieties having considerable resistance to early blight disease have been released for commercial use. However, completely resistant varieties still does not exist. Young plants are relatively resistant to the disease and susceptibility increases at maturity after fruit initiation.

Cultural practices: Good cultural practices play an important role in maintaining plants health while reducing the losses caused by early blight disease below economic threshold level. Transplant tomato seedlings on beds covered with plastic mulch which not only maintain soil moisture, reduce evapotranspiration and maintain low relative humidity in microclimate of crop canopy. Field sanitation plays a key role in reducing the initial inoculum of the pathogen as pathogen thrives on infected plant debris. Infected plant debris and decaying fruits should be removed from the field. Collateral hosts of solanaceous family viz., weeds and volunteer plants should be removed from the field prior to planting of crop. Crop rotation with non-host crop also reduces inoculum buildup. Irrigation should be avoided during cloudy weather or in the evening. Soil fertility plays an important role in disease management. The disease occurs in nitrogen deficient soil towards the maturity stage of the crop. Thus, optimum nutrition level in soil reduces plant stress and enhances plant health.

Chemical control: Fungicides having protectant and curative properties are effective in early blight disease management. Fungicides such as fluxapyroxad, flupyram and penthiopyrad can be effectively controlled the disease. Timing of application relative to environmental conditions and potential for disease development is crucial. Periodical foliar spray of azoxystrobin 23% SC@0.1% alternated with copper oxychloride 50%WP @0.25% and mancozeb 75% WG @0.2% at 8 days interval and spray must be started at disease initiation.

Other practices

- Field sanitation by plucking the lower leaves and burning of infected crop debris.
- Summer ploughing to increase the desiccation of pathogen and infected plant parts.
- Minimize relative humidity in plant canopy for preventing the infection.

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Name of disease: LATE BLIGHT

Geographical origin, distribution and crop losses: Late blight is the disease of historical significance as it caused Irish potato famine in 1845 and attacks both tomato and potato crop. The pathogen *Phytophthora infestans* probably originated from central Mexico. The disease was introduced into United States from Mexico and it further spread to North America and Europe. During subsequent decades, the disease appeared throughout the world and was widely distributed in the twentieth century. *Phytophthora infestans* is known to attack several plant species belonging to family *Solanaceae* and can cause complete devastation of tomato and potato crop. In 2009, the late blight disease appeared in epidemic form in US caused due to rapid dissemination of pathogen on infected tomato transplants (Hu *et al.*, 2012).

Symptoms: The disease is caused by *Phytophthora infestans* and is more common in North West states. But it also appears in Uttar Pradesh and Bihar in the months of December-January. Symptoms on leaves, twigs (**Fig 4**) and fruits are more common. Blight appears on foliage as light-pale green water soaked dead area. The lesions are wet in morning and dried, shriveled in daylight. It enlarges rapidly until entire leaves are

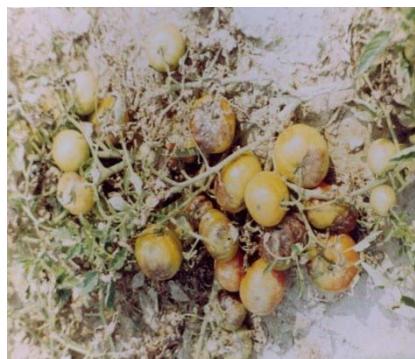


Fig. 4 Symptoms of late blight on twigs and tomato fruits



Fig. 5 Symptoms of late blight on twigs of tomato

killed and exposing all the fruits for infection (Fig 5). In moist and cloudy weather, a downy white fungus develops near the outer margin of lesion on underside of the leaves. Green and ripe fruit affected by the blight appeared as greenish brown and internal tissues become spongy with bad odor.

Causal organism: *Phytophthora infestans*

Key Characteristics: The pathogen *Phytophthora infestans* produces coenocytic, multinucleate mycelium although septation is seen in old cultures *in vitro*. The asexual spores known as sporangia are borne singly at the tip of alternately branched sporangiophores. The characteristic feature includes alternately branched sporangiophores with swelling at the point of attachment of sporangia. The sporangia produced are hyaline, lemon shaped with a distinct papilla at the distal end. The pathogen is heterothallic in nature as it requires A1 and A2 compatibility types for production of oospores which is rare.

Disease cycle and favourable conditions

The fungus *Phytophthora infestans* thrives on infected plants and tomato fruit as dormant mycelium. The volunteer tomato plants, infected fruits left in the field during harvest acts as source of primary inoculum. The secondary spread of the disease occurs through air borne conidia.

The growth of pathogen is favoured under cool, wet and cloudy weather conditions as clouds protect the spores from UV radiation exposure and high moisture content on the leaf surface allows germination of spores and subsequent infection. Optimum temperature of 60-80°F and high relative humidity (90%) are perquisite for disease initiation. The disease can also appear under warm day temperature (95°F) with moderate night temperatures (60-75°F). Sporangia may germinate directly via germ tube development at 70-79°F. At lower temperature, below 65°F sporangia germinates by production of zoospores. Zoospores require moisture for swimming and dispersal and capable of initiating new infection.

Management

Crop rotation: Rotation of tomato crops with non-host crops for duration of two to three years is essential for management of late blight disease.

Elimination of overwintering inoculums: Field sanitation is the preliminary step in controlling tomato late blight disease. The infected plants, plant debris, fruits and volunteer plants should be removed and destroyed to reduce the inoculum density.

Scouting for disease: Regular scouting of fields is essential for the growers to detect early disease onset and to prevent disease occurrence in severe form. It alerts the growers of potential disease outbreak.

Fungicides application: Application of fungicides is the last resort for management of late blight disease in humid areas. Contact fungicides are more effective in disease management without development of resistant pathotypes. Systemic fungicides such as cymoxanil, fluopicolide, dimethomorph etc. may also offer post-infection control. Periodical foliar spray in cloudy, cold and drizzling weather of azoxystrobin 23% SC@0.1% alternated with copper oxychloride 50%WP and @0.25% mancozeb 75% WG @0.2% at 7 days interval. One spray of cymoxanil 8%+ mancozeb 64%WP @0.2% alternated with azoxystrobin 11.5% + mancozeb 30% WP @0.15% or metalaxyl 0.25% at disease initiation.

- Always use healthy and certified seeds collected from disease free area.
- Infected crop debris and fruits must be collected from the field and burnt.
- Staking of plant reduces incidence of *Phytophthora* diseases in tomato.

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Name of disease: COLLAR ROT

Geographical origin, distribution and crop losses: The collar rot disease is one of the major threats in tomato cultivation. The disease is distributed in tropical and sub-tropical regions and warm temperate regions of Central, North, and South America, Australia, Southern Europe, Africa, Asia, and Hawaii. It occurs in diverse soils and has a very wide host range. Thiribhuvanamala *et al.* (1999) reported that the disease causes 30 per cent yield loss in tomato crop. The first report of this fungus *Sclerotium rolfsii* Sacc dates back to 1892 with Peter Henry Rolfs' discovery of the organism in association with tomato blight in Florida. After that different publications on this pathogen support evidence of its worldwide distribution.

Symptoms: The first symptom of disease is observed as soft tissue necrosis of bark of the stem near soil line. White, cottony and silvery mycelium growth is clearly visible on the affected portion. Fungal growth is also found just below the soil surface. Later on, white to light brown mustard like sclerotia is observed on the same portion. Progressive dropping and yellowing followed by wilting of the entire plant is observed (Fig. 6). Sometime plants collapse soon after infection. The disease is soil borne where pathogen survives in the form of sclerotia.

Causal organism: *Sclerotium rolfsii* Sacc

Key characteristics: *S. rolfsii* is necrotrophic, soil borne fungal phytopathogen that produces abundant white mycelium on infected plants and in culture. The fungus is characterized by the production of small, tan to dark brown or black spherical sclerotia of average diameter of 0.6 mm comprising three layers – outer rind, middle cortex and inner medulla. Mature sclerotia are hard, slightly pitted, and have a distinct rind. Although most sclerotia are spherical, some are slightly flattened. *S. rolfsii* does not form asexual fruiting structures or spores. The teliomorph of *S. rolfsii* (*Athelia rolfsii*) is rarely observed on hosts or in culture.

Disease cycle and Favorable conditions

The collar rot disease is typically a seed-bed disease which attacks young, tender plants in the seed bed that ultimately carries the disease to the main field. The infection is favored by high moisture, high temperature, high humidity, and moderate to heavy rainfall. The temperature requirement for hyphal extension and dry weight production of *S. rolfsii* is 8-40°C and maximum growth and scleria formation occur at 27-30°C. The fungus can survive in soil for more than four years in the form of dormant sclerotia. Due to its extensive host range and sclerotial survival in soil for a long time makes it very difficult to control.



Fig. 6. Rotting of stem due to collar rot caused by *Sclerotium rolfsii*

Management

Cultural method: Ensuring that plant residues have well decomposed before planting is important to manage the collar rot disease. Deep ploughing of soil to bury host debris and sclerotia to reduce inoculum levels in soil are found effective for the management of the disease. Crop rotation for 2-3 years with non-susceptible crops such as maize and small grains can reduce the disease. As a common cultural practice, removal of infected plants is an important aspect of Sclerotial disease management. *S. rolfsii* usually causes infection at the lower stem portion of the plant, and if the infected plants are allowed to remain in the field, these plants will serve as a continuous source of inoculum. That is why removal of the infected plant is necessary.

Biological method: In organic tomato cultivation, mulching by transparent polythene + soil application of Neem Seed Cake @ 150 kg/ha + soil application of *Trichoderma harzianum* @ 2.5 kg/ha followed by mulching + gypsum @ 4 tons/ ha. + spraying of *Trichoderma harzianum* @ 2.5 kg/ha can be effective for the management of the disease.

Chemical method: Seedling root dip treatment with tebuconazole (0.05%) and soil drenching with tebuconazole (0.05%) can be applied for the management of the collar rot disease of tomato.

Other practices:

- Use crop rotation with cereals, maize, sorghum and millets.
- Remove all the weeds from field.
- Avoid use of infested farmyard manure, soil and compost in the field.
- Irrigate the soil in summer and plough the field to destroy germinating sclerotia.
- Green manuring followed by application of *Trichoderma* formulation @5kg/ha within a week of ploughing.
- Seedlings dip in solution of *Trichoderma* @1% for 10 minutes.
- Avoid dense planting and maintain proper aeration near collar region.

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Name of disease: PSEUDOCERCOSPORE LEAF BLIGHT

Geographical origin, distribution and crop losses: This disease was first time observed in Uttar Pradesh during 2000-2001 in winter season in some of exotic lines. The pathogen was identified as *Pseudocercospora fuligena*. It caused severe leaf blight in tomato.

Symptoms: Black to olivaceous black, sooty growth of fungus are observed on the upper surface of the leaves (Fig. 7). The corresponding lower portion of leaves was light yellow to tan coloured. Leaves defoliated soon after infection. Symptoms were also observed in petioles, calyx and stem. This disease may create problem for tomato in future.

Causal organism: *Pseudocercospora fuligena*

Key characteristics: Characteristics of the associated fungi were fascicles amphigenous, predominantly hypophyllous, effuse, stromata absent or substomatal. Conidiophores pale olivaceous to olivaceous brown, 9-16 per fascicle, 20-39 x 3.5-5.0 µm, 1-2 septate, conidiogenous loci inconspicuous. Conidia obclavate to cylindric-obclavate, rounded to obtuse at the apex, olivaceous to subhyaline, obconic to rounded at the base, 29-110 x 2.5-5.0 µm and 3-10 septate, hila unthickened, not darkened.



Fig. 7. Symptoms of *Pseudocercospora* leaf blight on tomato leaves

Disease cycle and favorable conditions: *Pseudocercospora fuligena* is more common in the warm, humid climate of the tropics or subtropics than in the midwest. Both diseases cause chlorotic (yellow) lesions which are visible on the upper side of the leaf. The pathogen overwinters in soil. The optimum temperature for leaf mold is 71° to 75° F, while the optimum for Cercospora leaf mold is 82° F.

Management

- Collect and burn the infected leaves.
- Clean out high tunnel tomatoes between crops.
- Carefully select exotic hybrids and varieties.
- In the field, practice crop rotation and till under the crop as soon as the last fruit is picked.
- Need based one or two sprays of zineb 75%WP @0.2% alternated with mancozeb 75%WP @0.25%.
- A floor covering that prevents infected leaves from entering the soil will help lessen disease severity.

Name of disease: WHITE ROT

Geographical origin, distribution and crop losses: White rot is also called as Sclerotinia blight and caused by an ubiquitous pathogen called *Sclerotinia sclerotiorum*. The disease is observed in December to January during low temperature, cloudy weather and high atmospheric humidity accompanied by soil moisture.

Symptoms: Infection starts during flowering stage. Soft, wet, water-soaked rotting of stem, leaves, fruits, flowers are observed followed by white fungal growth over it. This mycelium develops honeydew stage soon after colonization. Later on, after drying the entire portion is converted in to compact mycelial mat followed by hard sclerotial bodies (Fig. 8). Fruits are severely affected where sclerotia formed inside and outside of the fruits.

Causal organism: The disease is caused by an ubiquitous pathogen called *Sclerotinia sclerotiorum*

Key characteristics: *Sclerotinia sclerotiorum* (Lib.) de Bary is the

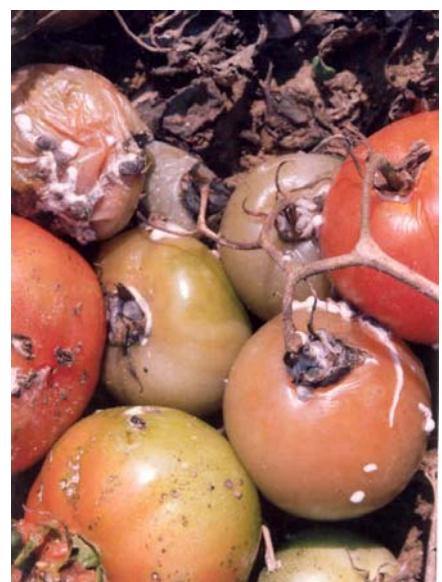


Fig. 8. Symptoms of white rot caused by *Sclerotinia sclerotiorum* on tomato

recognized name of the fungus causing the disease. Mycelium of the fungus is hyaline, much branched consisting of closely septate large hyphae which are both intra and inter cellular. The hyphae are 9-18 µm broad and filled with dense protoplasm. The fungus does not produce true conidia. Micro conidia (spermatia) may be produced during shortage of food supply on the disc of over mature apothecia. After completion of vegetative growth, the hyphae with thick granular protoplasm and short cells collect in small dense masses which gradually become sclerotia. Sclerotia are initially pink in color and turns dark brown to black on maturity with a dimension of 2.5-6 mm (Singh, 2004).

Disease cycle and favorable conditions: The fungus survives through sclerotia on the soil surface or in the crop debris or as admixture of seed during the off season. The primary infection takes place by ascospore produced from germination of these sclerotia. Wind borne ascospores, soil in the rhizosphere region of seedlings, farm machineries, animals and humans are the primary means of spread of the disease. Farm animal feed on the contaminated crop along with the sclerotia may also spread the disease by means of dung (Alkem and Melouk, 1990). The sclerotia can also undergo a hyphal or eruptive mycelial germination. In hyphal germination, production of a few short hyphal strands are observed. These hyphal strands can colonize on dead plant materials which come in contact with them. Using the colonized material as food source, the fungus can infect healthy host tissue. In eruptive mycelial germination, production of a massive and dense mycelium is observed. The mycelium can directly infect healthy host tissue. In both the process, more sclerotia are produced within a short period after infection, thus completing the cycle. Epidemics are initiated by ascospore developed from sclerotia during mid-winter. Clarkson *et al.*, (2003) have reported that release of ascospores occur in both saturated air (90-95% RH) and at 65-75% RH. The favorable temperature for germination of ascospore is 15-30°C. However, 100% germination is observed in the temperature of 20-25°C.

Management

Cultural:

- Seed must be free from sclerotia and seed infection. If sclerotia are observed in the seed lot, these should be removed with floatation. Seed borne mycelium can be inactivated by seed treatment with fungicide like iprodione + thiram. Seed treatment can also be carried out with the biocontrol agent like *Trichoderma harzianum* or *T. viride*.
- Soil borne inoculum in form of sclerotia should be deactivated with help of soil drenching fungicides. Sclerotia bearing plant parts should be destroyed and burnt immediately. Burying the sclerotia deep into the soil by ploughing at least for 30 weeks ensure destruction of them. The soil should be kept weed free as most of the common weeds are susceptible to this disease and harbor Sclerotinia fungi to build up to high levels in the soil.
- Avoid excessively high plant populations and narrow row spacing
- Crop rotation and no tillage is the most useful combination of treatments to reduce primary inoculum.
- There is a limitation to develop commercial plant resistance against Sclerotinia fungi. Disease avoidance is the most suitable technique. Reduction of the population of sclerotia surviving from one crop to the next is the essential to get rid of from this destructive disease.
- Cut the infected plant parts along with some healthy portion in morning and carefully collect in polythene to avoid falling of sclerotia in the field. Burn all these materials away from field.

Biological:

- Destruction of sclerotia by antagonistic fungi, bacteria and soil amoeba is also reported. The fungal parasite of sclerotia, *Sporodesmium sclerotivorum* and *Coniothyrium minitans* have also the potential to keep the numbers of inoculum in lower level.
- Spraying of ziram, ferbam or systemic fungicides of benzimidazole group have proved effective in checking spread of the disease. Tetrasodium thiocarbamate and the dicarboximide fungicide such as procymidone, vinclozolin and iprodione are highly effective against *Sclerotinia* (Singh, 2004).

Chemical:

- Foliar spray of mancozeb @0.2% at flowering stage followed by spray of copper oxychloride 50%WP @0.25% in cool, cloudy and humid weather.

Name of disease: FRUIT ROTS

Geographical origin, distribution and crop losses: Fruit rot or Buckeye rot is caused by three species pathogen *Phytophthora*: *P. nicotianae* var. *parasitica*, *P. capsici*, and *P. drechsleri*. It is a fungus that thrives in warm, wet conditions and lives in the soil. It is characterized by a bull's eye pattern of dark brown rotting on the tomato fruit, and affects fruit that is close to, or lying on the soil. The easiest management is to keep the plant out of contact with the soil, although other chemical methods can be very effective. This disease commonly occurs in the southeast and south-central areas of the United States. The disease has affected a large portion of crop yield in the United States as well as India. The relatively small genome size of *Phytophthora parasitica* compared to *Phytophthora infestans* gives researchers the unique ability to further examine its ability to cause disease. Besides *Phytophthora* other pathogen like *Colletotrichum coccodes* is also involved in this disease.

Symptoms: Symptom of Myrothecium fruit rot on green as well as ripe fruit appeared as water-soaked rotting with prominent concentric zonation. White to black numerous sporodochia was observed on each ring of rotting. Rhizoctonia fruit rot is most serious disease of kharif tomato. Symptoms appear as rhythmic rotting of fruits coming with the soil contact. Rotting is immediately followed by cracking and fungal growth over it (Fig 9). Buckeye fruit rot caused by *Phytophthora parasitica* was observed mostly in green fruits. Symptoms of ripe fruit rot caused by *Colletotrichum coccodes* were first time observed in 2002-2003 as black, dry and corky rotting with acervuli over it (Fig. 10). Sclerotium fruit rot is very clear due to white fungal mycelium and mustard grain like sclerotia over fruits (Fig 11). Sclerotinia fruit rot is having bigger and irregular sclerotia over it. Generally lower portion of fruits are infected which are coming in soil contact (Fig 12).



Fig 9-12. Various symptoms of fruit rot infections

Causal organism: Buckeye fruit rot caused by *Phytophthora parasitica* and ripe fruit rot caused by *Colletotrichum coccodes*

Key characteristics: *Phytophthora parasitica* is a soilborne pathogen with a wide range of host plants and represents most species in the genus *Phytophthora*, being capable of infecting over 72 plant genera.

Disease cycle and favorable conditions:

Buckeye rot of tomato is soil-borne and therefore affects fruit lying on, or close to, the soil. The fungi are spread by surface water, spattering rain, and furrow irrigation. While it can sexually reproduce through the production of oospores, its primary form of reproduction is by asexually producing sporangia. These sporangia are found at the tips of sporangiophores that emerge through the stomates. Sporangia release zoospores which require water for transport. Zoospores swim in water droplets until they encyst and infect the fruit. Symptoms, beginning with fruit rot, may form within 24 hours. Chlamydospores are the pathogen's resting structure, which allows the disease to survive and overwinter in the soil. The chlamydospores germinate in soil or decaying debris. Through these chlamydospores, the pathogen begins the disease cycle again. The combination of asexual and sexual spores makes this disease polycyclic, therefore it can infect multiple times throughout a season.

Management

Cultural:

- Maintain proper soil moisture.
- Avoid the contact of fruit from soil by staking of plant.
- Provide proper drainage in the field.

Chemical control:

- Apply fungicides containing mefenoxam to the soil surface under the vines around 4–8 weeks prior to harvest.
- Soil should be fumigated before anything else is planted in previously-infected soil.

Biological:

Green manuring followed by soil application of *Trichoderma* @5 kg/ha in soil is very effective in checking most of the fruit rotting. Collect affected fruit and burn them to reduce primary inoculum.

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Name of disease: GREY LEAF SPOT

Geographical origin, distribution and crop losses: Stemphylium gray leaf spot of tomato is distributed worldwide including the United States, particularly in humid tropical and subtropical regions. Currently, this disease remains one of the most destructive diseases of tomato in the southeastern United States. The disease mainly attacks tomato leaves at all growth stages from seedling to harvest. On susceptible cultivars, the disease can cause significant early defoliation, which leads tomato fruit prone to sunscald. The pathogen also infects pepper, eggplant, potato, and some solanaceous weeds. *Stemphylium solani* is found throughout the world. It was first described by George Weber in 1930 in Florida, United States. It has since been reported in Brazil, Venezuela, India, South Africa, Spain, Australia, Egypt, and China.



Fig. 13. Symptoms of grey leaf spots on tomato leaves

Symptoms: Small circular dark brown necrotic, numerous spots appeared all over the leaf lamina that is uniformly distributed on entire foliage (Fig. 13). Sometime clear narrow yellow halo is visible around the spots. Usually these spots are isolated but in later stage they coalesced to each other. Severely infected older leaves blighted and fall. Small elongated to circular lesions appeared on stem after brightening of leaves.

Causal organism: This disease is very common during winter and caused by *Stemphylium solani*.

Key characteristics: Colonies are with typical septate mycelium with the young hyphae subhyaline and gradually became greyish green to dark brown with age. Conidiophores are subhyaline to light brown, 3- to 10-septate, up to 200 µm in length, and 4 to 7 µm in width, with apical cell slightly to distinctly swollen, bearing a single spore at the apex. Conidia were muriform, mostly oblong to ovoid, but occasionally nearly globose, subhyaline to variant shades of brown, mostly constricted at the median septum, 22.6 ± 6.22 (11.9 to 36.9) µm in length, and 15.1 ± 2.85 (8.3 to 22.6) µm in width, with 1 to 8 transverse and 0 to 5 longitudinal septa.

Disease cycle and favourable conditions: Three fungal species in the genus *Stemphylium*, *S. lycopersici*, *S. botryosum* sp. *lycopersici* and *S. solani*, have been reported to cause gray leaf spot of tomato. The pathogen survives in plant debris and can be seedborne. In the region where tomato is grown throughout the year, the pathogen can remain viable on living tomato plants. An epidemic of the disease may begin in infested seedbeds. Infected tomato seedlings, plant debris and volunteer pepper, eggplant and other wild solanaceous can serve as inoculum sources for disease epidemics in fields. Fungal spores can be dispersed by wind or air circulation, and by splashing water resulted from rainfall and overhead irrigation. Spore germination and infection require high humidity or free water on leaf surfaces. The optimal temperature for the disease development is 77 °F. So, the disease is favored by warm and wet weather conditions.

Management

Cultural:

- Use of resistant varieties is an effective and economic way to control this disease.
- Removal and burning of all crop debris after senescence of the crop.
- Periodical monitoring of the crop for initiation of disease from last fortnight of December to first week of January.
- Rotate tomato with non-solanaceous crops for 2-3 years.

Chemical:

- Foliar spray of either zineb 75%WP @0.2% alternated with mancozeb 75%WP @0.25%.

Name of disease: BACTERIAL SPOT

Geographical origin, distribution and crop losses: The disease is prevalent throughout the state. Kharif crop of tomato is severely affected by the bacteria with an average incidence of 35-40% every year. Yield loss is recorded more than 40% in kharif tomato. Disease continues from July to October. Prominent yellow halo is observed around the spot.

Symptoms: Small, dark spots appear on leaflets and stems of seedlings transplanted crop (Fig. 14). Numerous spots coalesced and cause severe burning of the leaves. The most conspicuous and damaging phase is infection of immature fruits. Small water-soaked spots appear on exposed surfaces of the green and turning red fruits. These are slightly raised and corky in appearance (Fig. 15).

Causal organism: Bacterial spot of tomato is caused by *Xanthomonas vesicatoria*.

Key characteristics: The bacteria that cause bacterial spot are called xanthomonads. Recent taxonomic studies have indicated that these bacteria belong to one of four groups: A, B, C and D. The original (and still valid), taxonomic name given to these bacteria was *Xanthomonas campestris* pv. *vesicatoria*, but recent work has indicated that each group may represent a separate species. It is a Gram-negative and rod-shaped. It causes symptoms throughout the above-ground portion of the plant including leaf spots, fruit spots and stem cankers. Since this bacterium cannot live in soil for more than a few weeks and survives as inoculum on plant debris, removal of dead plant material and chemical applications to living plants are considered effective control mechanisms.

Disease cycle and favourable conditions:

Xanthomonas campestris pv. *vesicatoria* survives on tomato and pepper plants, seeds, and debris from infected plants as it cannot live in the soil for more than a few weeks. The bacterium can also be found in association with wheat roots and some weed species which are both considered sources of inoculum as well as diseased tomato and pepper plants. In cold climates, *Xanthomonas campestris* pv. *vesicatoria* infection is mostly caused by contaminated seed material, both on and inside of seeds. If it survives on seeds, it will infect the cotyledons of the growing plant as it emerges from the seed coat. Internally infected seeds will produce diseased plants from the point of germination. Systemic symptoms such as wilting, yellowing, and dwarfing are not typical of plants infected at the point of germination. However, foliage loss can happen when localized symptoms on leaves become severe. If the bacterium survives on debris, it may infect healthy plants through stomata as well as wounds on leaves and fruit. It is spread by direct contact of plants with debris, human movement of the bacteria from debris to plants, and can easily travel from debris to healthy plants through saturated soils via water movement. Once infected, plants begin to develop lesions on the leaves as well as fruit, becoming inoculum sources for further infection.

Disease appeared in rainy season and continued up to fruit initiation stage. The disease is prevalent in warm humid weather when RH is more than 90% and temperature is between 25 and 35°C.



Fig. 14. Small dark spots on tomato leaflets



Fig. 15: Raised corky surface of tomato fruits

Management

Cultural:

- Summer ploughing to desiccate the bacteria and host.
- Soil solarization in nursery bed to avoid seedling infection.
- Rotation of nursery seedbed and main field.
- Seed should be collected from disease free plants.

Chemical:

- Foliar spray of copper oxychloride @0.25% in afternoon.

Name of disease: BACTERIAL SPECK

Geographical origin, distribution and crop losses: This disease is of moderate economic importance to tomato production under greenhouse and field conditions.

Symptoms: It is characterized by absence of chlorotic halo around spot, crusty and comparatively larger spots (Fig 16). Sometime yellow halo is observed in this infected plants. The spots are necrotic, circular to roughly circular. As the fruit approaches the pink stage, the tissue around the spot retains its green colour longer than normal. Small black spots appear on leaf, petiole, pedicel, and peduncle while irregular, elongated lesions on stem.

Bacterial speck lesions may occur anywhere on the foliage, stems or fruit. Symptoms are very difficult to visually distinguish from bacterial spot and can be confused with young, early blight lesions. On leaves, symptoms appear as black specks, usually no more than 2 mm in diameter, which are usually surrounded by a yellow halo. Speck lesions sometimes cause distortion of the leaf, as the infection restricts the expansion of leaf tissue. Lesions are often concentrated near leaf edges, and in some cases, leaf margin burn resembling bacterial canker may occur. When numerous, lesions may coalesce, and entire leaflets may die. Severely infected seedlings may become stunted. Only green fruit less than 3 cm in diameter is susceptible to infection by the bacterial speck pathogen. Small (less than 1-3 mm), slightly raised black specks develop and are often surrounded by a narrow green to yellow halo. Lesions are usually superficial and can be scraped off with a fingernail. Red fruit are not susceptible to infection, likely due to a lack of entry points for bacteria; fruit hairs, which may break and allow bacteria to enter, are only present on young fruit. On fruit previously infected, black lesions remain after ripening.

Causal organism: The disease is almost similar to bacterial spot but caused by *Pseudomonas syringae* pv. *tomato*.

Key characteristics: *Pseudomonas syringae* is a rod-shaped, Gram-negative bacterium with polar flagella. As a plant pathogen, it can infect a wide range of species, and exists as over 50 different pathovars, all of which are available to researchers from international culture collections such as the NCPPB, ICMP, and others. *Pseudomonas syringae* is a member of the genus *Pseudomonas*, and based on 16S rRNA analysis, it has been placed in the *P. syringae* group. It is named after the lilac tree (*Syringa vulgaris*), from which it was first isolated.



Fig. 16. Symptoms of bacterial speck on tomato leaves

Disease cycle and favourable conditions: Bacterial speck is prevalent in November-December and favoured by cool, moist, weather and temperature range between 15 and 25°C. Generally, disease is observed in winter season. *Pseudomonas syringae* overwinters on infected plant tissues such as regions of necrosis or gummosis (sap oozing from wounds on the tree) but can also overwinter in healthy looking plant tissues. In the spring, water from rain or other sources will wash the bacteria onto leaves/blossoms where it will grow and survive throughout the summer. This is the epiphyte phase of *P. syringae*'s life cycle where it will multiply and spread but will not cause a disease. Once it enters the plant through a leaf's stomata or necrotic spots on either leaves or woody tissue then the disease will start. The pathogen will then exploit and grow in intercellular space causing the leaf spots and cankers. *P. syringae* can also survive in temperatures slightly below freezing. These below freezing temperatures increase the severity of infection within trees like sour cherry, apricot, and peach. Diseases caused by *P. syringae* tend to be favoured by wet, cool conditions optimum temperatures for disease tend to be around 12–25 °C (54–77 °F), although this can vary according to the pathovar involved. The bacteria tend to be seed-borne, and are dispersed between plants by rain splash.

Management

Cultural:

- Summer ploughing to desiccate the bacteria and host.
- Soil solarization in nursery bed to avoid seedling infection.
- Rotation of nursery seedbed and main field.
- Seed should be collected from disease free plants.

Biological:

- Spray *Bacillus subtilis* or *Pseudomonas fluorescens* formulation @4g per liter water.

Chemical:

- One spray of copper oxychloride @0.3%, after 10 days of antibiotic application.

Name of disease: LEAF CURL COMPLEX

Geographical origin, distribution and crop losses: Average incidence of leaf curl complex in kharif tomato is 60-65% every year. Virus is transmitted by white fly as well as by mechanical injury. Tomato yellow leaf curl virus (TYLCV) seriously impacts tomato production throughout tropical and sub-tropical regions of the world. It has a broad geographical distribution and continues to spread to new regions in the Indian and Pacific Oceans including Australia, New Caledonia and Mauritius.

Symptoms: Disease appear in severe forms during the month of September to November. Leaves show downward rolling, curling, twisting and chlorosis. Plants show stunting, shortening of internodes, bushy appearance, no flowering and fruiting at later stage of virus infection. Sometime leaves show light and dark mottle symptoms. Since begomovirus and TMV both viruses are involved in most of the infection, therefore showed symptoms of leaf curl as well as mosaic are observed on infected plants (Fig 17).

Causal organism: Begomovirus and TMV

Key characteristics: *Begomovirus* is a genus of family *Geminiviridae*. They are plant viruses have a very wide host range, infecting dicotyledonous plants. Worldwide

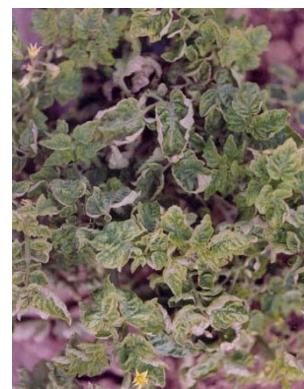


Fig. 17. Symptoms of tomato leaf curl disease

they are responsible for a considerable amount of economic damage to many important crops such as tomatoes, beans, squash, cassava and cotton. There are 445 species. *Begomovirus* is a genus of viruses, in the family *Geminiviridae*.

Tobacco mosaic virus has a rod-like appearance. Its capsid is made from 2130 molecules of coat protein and one molecule of genomic single strand RNA, 6400 bases long. The coat protein self-assembles into the rod-like helical structure (16.3 proteins per helix turn) around the RNA, which forms a hairpin loop structure. The protein monomer consists of 158 amino acids which are assembled into four main alpha-helices, which are joined by a prominent loop proximal to the axis of the virion. Virions are ~300 nm in length and ~18 nm in diameter. Negatively stained electron microphotographs show a distinct inner channel of radius ~2 nm.

Disease cycle and favorable conditions: The virus is obligately transmitted by an insect vector, whitefly *Bemisia tabaci* or other whiteflies. This vector allows rapid and efficient propagation of the virus because it is an indiscriminate feeder. The vector transmits in a persistent, circulative, non-propagative manner.

Management

Cultural:

- Removal of weed host from field and surrounding areas.
- Nursery should be grown in nylon net to check the white fly infestation.
- Avoid early crop and transplanting should not be done before September.
- Use barrier crop of taller non-host crop like maize, bajra and sorghum.
- Collect healthy seeds from disease free plants.
- Avoid mechanical injury during intercultural operations.
- Rouging of infected plants soon after infection at initial stage of growth.

Chemical:

- Root dipping in imidacloprid solution @0.4 ml per liter of water for one hour during transplanting of the seedlings.
- Seed treatment with 10% tri sodium phosphate solution for 25 minutes.
- Periodical foliar sprays of imidacloprid 17.8% SL@0.4 ml per liter of water alternated with thiamethoxam 25.0% WG @0.3 gram/liter of water at 10 days interval up to flower setting.

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Name of disease: TOSPOVIRUS

Geographical origin, distribution and crop losses: Tomato spotted wilt virus (TSWV), the type member of the tospoviruses is found worldwide in temperate regions in association with its thrips vector. The wide host-range of TSWV and its thrips vector is consistent with the geographic distribution. Other tospoviruses have more well-defined distribution. For example, GBNV, WBNV, and WSMoV, that are transmitted by *Thrips palmi*, a thrips species found only in the subtropics are only known to occur in Southeast Asia. Another anomaly is INSV. While INSV is reported to occur around the world, it is almost entirely limited to greenhouse-grown floral crops.

Symptom: The symptoms of tospoviruses vary greatly with the host affected, plant organ affected, and age of plant or organ at the time of infection. In general, however, tospovirus symptoms appear as chlorotic or necrotic rings, lines, or spots on leaves, stems, and fruits; necrotic streaks on stems; bronzing, curling, and wilting of leaves; rings, necrotic spots, and malformations on fruits; stunting and necrosis of parts or whole plants; and, generally, greatly reduced yields.

Causal agent: Tomato spotted wilt virus (TSWV)

Key Characteristics: The tospovirus particles are spherical, about 80 to 110 nanometers in diameter, and are enveloped with a membrane. Each virus particle contains four types of proteins: two glycoproteins (78 and 58K) partially embedded in the membrane and forming two types of external projections ; a 29K protein associated with the three viral RNAs within the particle and forming three pseudocircular nucleoprotein structures; and a few molecules of a large (330K) protein that is thought to be the virus replicase.

Disease cycle and favourable conditions: Tospoviruses are transmitted by at least seven species of thrips. These include the western flower thrips (*Frankliniella occidentalis*), tobacco thrips (*F. fusca*), common blossom thrips (*F. schultzei*), onion thrips (*Thrips tabaci*), and melon thrips (*T. palni*). Tospoviruses can be acquired from infected plants by thrips larvae but not by adult thrips. Once a larva acquires the virus, however, usually after feeding on an infected leaf for 30 minutes or more, it retains the virus through molting, pupation, and emergence so that the emerging adult thrips is viruliferous and can transmit the virus to healthy plants for the rest of its life. Inoculation feeding periods must be 30 minutes or longer. Fortunately, adult thrips, once alighting, do not move from plant to plant as much as aphids do and so transmission of tospoviruses is not as explosive as some of the aphid-borne viruses.

Tospoviruses overwinter on their perennial or biennial hosts from which their thrips vectors transmit them to healthy plants. The virus spreads into phloem and parenchyma cells and multiplies in their cytoplasm. Virus particles appear to form in densely staining patches of cytoplasm (viroplasms), possibly by budding, and release from regions of two parallel membranes. Mature individual particles or groups of particles are always surrounded by irregularly shaped membranous cisternae.

Management:

Cultural:

- Avoid cultivation of susceptible crops.
- Only tospovirus-free transplants should be planted.
- Plants should be monitored for thrips vectors and should be treated with insecticides to keep thrips populations to a minimum
- Rouging of infected plants may help.

Name of disease: TOMATO BIG BUD

Geographical origin, distribution and crop losses: Tomato big bud has been emerging as a major phytoplasma associated disease in different states of India and farmers and seed companies are facing serious losses to the crop and income every year. Incidence of the disease varied between 2.0 and 40%, depending on the region and weather conditions. An infected plant formed on average 70% less fruit than a healthy plant.

Exact information on geographical origin is uncertain. There are many other countries where tomato big bud has been reported, for instance, China, India, and other Asian countries, South Africa, and the USA, but the strains have not been reported. Tomato big bud caused by 16SrII group phytoplasma was first recorded in Australia, it is reported from many Pacific Islands countries e.g. New Caledonia from tomato. Big bud of unknown phytoplasma strain has also been recorded in tomato from Fiji.

Symptoms: Severe phyllody and big bud symptoms are the common symptoms in phytoplasma infected plants (Fig. 18-22). All the flower buds are malformed and there is no fruit production at all (Rao, 2021). The disease is reported to be associated with peanut witches' broom (*Candidatus Phytoplasma australasia* related strain; 16SrII-D subgroup). The most striking symptom of tomato big bud is the large, swollen green buds that fail to develop normally and do not set fruit. Apical stems are thick and assume an upright growth habit. Infected plants appear bushy because of shortened internodes and small leaves. Leaves are distorted and yellow-green.



Fig. 18-22: Tomato big bud, flower malformation and shoot proliferation symptoms caused by phytoplasmas in tomato

Causal agent: Phytoplasmas are cell wall-less bacteria-like microorganisms, which are generally transmitted by leafhoppers and can inhabit and propagate in both plant and insect vectors. Phytoplasmas are cell wall-less bacteria-like microorganisms, which are generally transmitted by leafhoppers and can inhabit and propagate in both plant and insect vectors. *Candidatus Phytoplasma aurantifolia*; strains has been identified in tomato throughout the world.

Disease cycle and favourable conditions: So far, the disease cycle and epidemiology of the disease is not known. Beet leafhopper transmitted viresence agent, a phytoplasma organism, is transmitted by the beet leafhopper, *Circulifer tenellus*. Spread of the phytoplasma is by leafhoppers. One leafhopper that spreads tomato big bud is *Orosius argentatus*, but it is possible that there are other species. This leafhopper lives and breeds on weeds and moves to tomato (and other crops) during dry times. Spread can be over long distances, during leafhopper migrations.

The phytoplasma survives in weeds, and in the bodies of the leafhoppers. Once leafhoppers become infected by feeding on a plant with phytoplasma, they remain infected for the entire life of insect.

Management:

Before planting:

- Remove any ‘volunteer’ tomato plants and weeds, especially those in the tomato family, from around nurseries and in and around field plots.
- Preferably, raise the tomato seedlings in a screenhouse or under an insect screen cover.
- Do not plant next to other susceptible crops, such as eggplant or capsicum.

After harvest:

- Collect the plants together with weeds around the planting and burn or bury them.

Chemical:

- Early diagnosis of the disease in the field and management of leafhoppers through spraying imidacloprid @ 3ml/10 lit water is effective in minimizing the disease incidence.

Reference:

Rao GP (2021) Our understanding about phytoplasma research scenario in India. Indian Phytopathol 74: 371–401. DOI: 10.1007/s42360-020-00303-1

Brinjal

Introduction

Brinjal (*Solanum melangena* L.) is available in variety of colors; purple, violet, green, white and in elongated or round shape. India, accounts for about 8.7 million MTs with an area of 0.53 million hectares under cultivation of brinjal. Brinjal is rich in fiber; people consume eggplant to facilitate proper bowel movement. Brinjal contains plenty of carbohydrate and 100g serving of raw brinjal contains 2.5g of carbohydrate, 2.5g of fiber, and 75kj of energy. It is a rich source of minerals like potassium, copper, magnesium and manganese and vitamins (B6, C, K, folate, niacin) and is low in saturated fats, cholesterol and sodium. People suffering from heart disease, high cholesterol and hypertension can eat eggplant in liberal quantity. Purple skinned eggplants have phenols which help our body to make use of the blood sugar. Phenols also help in inhibiting an enzyme causing high blood pressure in the body. A handful of bacterial and fungal diseases can strike your eggplant patch, causing blemished fruits, marred foliage, decreased production and sometimes plant death. Damping off (*Pythium* spp., *Phytophthora* spp., *Rhizoctonia* spp.), Phomopsis Blight (*Phomopsis vexans*), leaf spot (*Cercospora melongenae*), Alternaria leaf spots (*Alternaria melongenae*), fruit rot (*Phytophthora nicotianae*), Verticillium Wilt (*Verticillium dahliae*) are major diseases of brinjal and cause significant losses in yield and quality of the produce.

Damping off

Geographical origin, distribution and crop losses: Damping off is a severe disease of solanaceous crops including eggplant in nursery. Several soil borne pathogens attack seed, seedling and transplant and thus cause disease in plants. The disease is not only responsible for the poor seed germination and stand of seedlings, but also carry the pathogens into the main field and affect the crop. The diseased seedlings are one of the major sources for rapid and efficient spread of disease in the field. Damping off disease spreads in nurseries and transplanted plants from initial infection points, causing large areas in which usually all the seedlings are killed. Many researchers reported severe losses caused by this disease.

Symptoms: Affected plants generally occur in scattered patches in nursery or in lower parts of the slope fields. In even slope fields, diseased plants are usually occurring randomly in the fields. Damping off may occur as pre or post emergence. In pre-emergence damping off, target of the pathogen is seed and on infection it fails to grow after sowing. On attack of the pathogen seeds soft, mushy and turn brown, and later decompose because of seed infection. In post-emergence damping-off, the seed germinate from underneath the soil, but dies soon. The affected portions mainly root, hypocotyls and crown of the plant, become pale brown, soft, water soaked and weaker than non-affected part. Infected stems collapse. Root rot and collar rot makes the plant stunted. Severity of the symptoms usually changes with the stage of the crop development. Pathogen takes time to establish in the host and thus infection by the pathogen(s) may occur much later after plant emerges; in this case, the infection is usually not severe, but the overall plant growth and yield production may be affected.

Roots are the most severely affected part of the plant may show wilt or rot in warm or windy weather. Severe root rotting results in nutrient deficiencies symptoms in the plant since nutrients may not be sufficiently available from the soil up through the plant. Since number of pathogens are responsible for identical symptoms, identification of respective pathogen after isolation is needed to confirm diagnosis.

Causal organism and Key characteristics: Damping off diseases is caused by number of *Pythium* spp. such as *Pythium aphanidermatum*, *P. butleri*, *P. inflatum*, *Phytophthora parasitica*, *Fusarium oxysporum*, *F. solani*, *Phomopsis vexans*, *Colletotrichum capsici*, *Rhizoctonia solani* and *Sclerotium rolfsii*. In *Pythium*, *P. aphanidermatum* and *P. butleri* are common species and mycelium of *P. aphanidermatum* is white, well developed, and woolly but not as abundant or vigorous as of *P. butleri* and less profusely. The hyphae are 2.8 to 7.5 µm in diameter, sporangiophore produces lobed, toruloid, branched. Encysted zoospores are 6.7-12.0 µm in size. Spherical, smooth oogonia are formed terminal on later hyphae and antheridia form monoclinous, intercalary or terminal. On each oogonia, 1-2 antheridia attach. Oospores are single aplerotic, moderately thick walled (17-19 µm).

Disease Cycle and Epidemiology: Seedlings during the initial three weeks after sowing are susceptible under the following predisposing factors: When planted in heavily infested soil or medium growth, overwatering or poor drainage, overcrowding of the seedling with poor ventilation, excessive doses of nitrogen, variable environmental conditions such as rainy, cloudy weather that results in prolonged soil moisture, low sunlight that prevents drying. These pathogenic fungi cause disease in a wide range of plants. These pathogens belong to similar genera of fungi with different environmental requirements for the disease development. For example, cool and wet climate favours *Pythium* and *Phytophthora*. Within *Pythium* spp., *P. ultimum* proliferate well under the low temperatures, while *P. aphanidermatum* active in high temperatures. Overwintering spores of these fungi can survive well in soil for a long period in the non-availability of host plants. favourable temperature for pre-emergence damping off is 20-25°C, while post emergence damping off was more severe at 40°C. High soil moisture and soil temperature accelerate development of the disease. These fungi colonize on the various plant debris. Clay soil favoured the most damping off disease's incidence caused by *P. aphanidermatum* and very less in sandy soil. Irrigation system in green houses contributes to fast spread of the *Pythium* spp. They form oospores and chlamydospores on decaying of plant roots which later survive for long period under adverse conditions and leads to subsequent infection. The disease incidence is enhanced, if nematodes are already present in nursery soil, which helps to cause injury to the roots for entry of the pathogen.

Disease management

Cultural practices: Soil solarization of irrigated soil for 30 days decreased pre and post occurrence of damping off disease in brinjal. Nursery sowing in the same bed year after year with frequent heavy irrigation should be avoided. Healthy brinjal seedling should be raised in the nursery bed with the field soil added with sand and farm yard manure.

Biological control: Seed treatment with native biological consortium *Pseudomonas* and *Azotobacter* species provides an enhanced level of protection compared to the native individual antagonistic bacteria to control pre and post emergence damping of brinjal. *Trichoderma harzianum* and *T. viride* formulations applied as seed treatment reduced damping off in brinjal and brinjal in the field and enhance the plant biomass under field conditions

Seed treatment with *Murray koenghii* (20% extract) showed better results in managing damping off. Inoculation of mycorrhizal fungus *Glomus fasciculatum* either simultaneously or 2 weeks before the inoculation with *P. aphanidermatum* reduced damping off and increased plant height in comparison to control. Neem cake extract, *Lantana camara* leaf extract, cow urine, *Sapium* sp., *Ligustrum nepalensis*, *Utrica parviflora*, *Eucalyptus* sp.

and azadirachtin were found very effective for the management of pre-emergence damping off while *Thuja compacta* and *Curcuma longa* were also found effective to control post emergence damping off.

Chemical control

Many researchers worked on seed treatment and soil sterilization with fungicides for management of this disease in the nursery and reported efficacy of soil fumigation with Metham, formalin and methyl bromide to reduce the population of soil borne fungi. Pre-sowing application of dazomet (basamid G @ 30 g/m²) was found effective in the management of damping off caused by *P. aphanidermatum*. Various fungicides such as cuprous oxide, MEMC, copper oxychloride, carbendazim, thiram carbendazim + thiram and carboxin have been recommended for the seed treatment to control the disease in the vegetables seed treatment with Metalaxyl 64% +Mancozeb 64% @ 2 g/kg seed alone or along with soil application of Metalaxyl 64% +Mancozeb 64% @ 0.2% twice, first immediately after germination and second at 20 days after germination resulted in the effective control of damping-off disease in the solanaceous crop nurseries.

Integrated management of disease:

Soil solarization with introduction of biocontrol agent *P. fluorescens* (PfT-8) and *T. harzianum* reduced the rhizosphere population of *Pythium* spp. by 86% and 82% respectively. Combination of soil solarization (30-40 days), organic amendments (FYM and poultry manure) and seed treatment with *T. viride* was found most effective approach for reducing pre and post emergence of damping off in brinjal. Consortial application of *P. fluorescens* (PfT-8) as seed treatment and soil application along with soil application of organic amendments such as FYM of poultry manure is needed which reduced disease incidence to 90% and improve plant health and increased rhizosphere population of bioagent.

References:

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Name of disease: PHOMOPSIS BLIGHT

Geographical origin, distribution and crop losses: Phomopsis blight is a disease of warmer region of the globe except Europe (in Romania it is present) and some African countries. This disease is native of South Asia, where brinjal or eggplant has been originated. The pathogen of Phomopsis blight overwinter in crop debris. The conidia spread through local transmission through the wind and rain splashes naturally. This disease effects the brinjal production severely, where it reduces 40 – 70% of the total yield.

Symptoms: Infected seeds cause damping off in the nursery. Leaves show clearly defined circular, light brown spots mostly in lower leaves. Center of the spots is slightly lighter in color. Old spots are having numerous black dots like pycnidia. These spots are papery, often crack and sometime shot hole. Sometime irregular, big lesions are also observed on leaves, particularly in cool and humid weather. Stem lesions are started from basal part near nodal region as gray dry rot with constriction. Bark partially dries but later entire twig dries. This leads to drying of few twigs or partial wilting of the same portion of plant. Old and dried twigs show several dots like erumpent pycnidia on the bark. Pale to light brown sunken spots develop on the old fruits. Individual spots expand and coalesce to cover most part of the fruits (**Fig 23**). Fruit rot is soft, spongy and with rhythmic growth. Mature, prominent and erumpent black pycnidia are found on inner circle of rot while

submerged conspicuous and immature pycnidia are found on the outer ring of rotting with concentric arrangement. Later, the infected fruit is completely rotted and mummified. The entire seeds of infected fruit are colonized by the pathogen and disease becomes internal seed borne.

Causal Organism: *Phomopsis vexans*

Key Characteristics: *Phomopsis* species have two types of conidia. Alpha-conidia are hyaline, fusiform, straight and usually biguttulate. Beta-conidia are hyaline, fusiform, straight or more often hamate, egutullate and aseptate. *Phomopsis* culture has two distinguished types of colonies. W-type colonies has surface view of white, aerial hyphae, scattered relatively large stroma, irregular pycnidial locules while reverse view is whitish and occasionally had pale pink, brown and or grey zones. G-type colonies has surface view of a few aerial hyphae, white to grey and formed abundant relatively small pycnidial stroma with irregular pycnidial locules and grey and brownish reverse view.

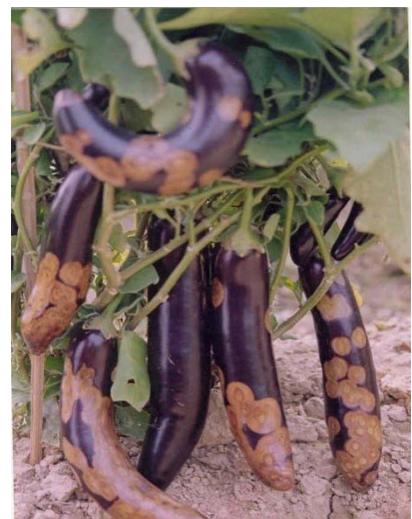


Fig. 23. Symptoms of *Phomopsis* blight on brinjal

Disease cycle and favorable conditions: *Phomopsis vexans* proliferate in hot and humid climate. Favorable or optimum temperature for germination of spores is 27°C whereas the formation of pycnidia is optimum at 30°C – 35°C and the favorable temperature for the growth of *Phomopsis vexans* is 28°C. The optimum relative humidity for the development of disease was 55%.

Management

- Seed should be collected from healthy fruits and disease-free field.
- Seed treatment with carbendazim @0.25%.
- Spray carbendazim @0.05% after 15 days of transplanting and during flower setting.
- Infected crop residue and fallen mummified fruits should be collected and burnt.

References:

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Name of disease: RHIZOCTONIA ROOT ROT

Geographical origin, distribution and crop losses: It is distributed worldwide and is prevalent in arid, sub-tropical and tropical climate, especially in the areas with low rainfall and high temperature. Generally early transplanted brinjal crop is much affected in the month of August-September. 15-20% disease incidence is recorded in farmers' field during rainy season.

Symptoms: Lesions starts on stem near collar region at or below the soil level and move downward into the roots. Initially bark become wet, soft with macerated tissue. Later on, drooping and wilting of the plant

observed (Fig 24). Disease is more common in poorly drained soil and the fields having prolonged excessive moisture.

Causal organism: *Rhizoctonia solani*

Key characteristics: *Rhizoctonia solani* is a common soil inhabitant which can survive many years in the absence of brinjal crop. *R. solani* can survive on crop debris and in soil as black to brownish resting structures (sclerotia) or as resting fungal mycelium (thread like material).

Disease cycle and favourable conditions: Damage to brinjal is dependent on many factors including soil moisture and temperatures, soil pH, herbicides used, fertility levels, and competition from other soil microorganisms. Disease development the maximum incidence was recorded in October at a maximum temperature of 32.7°C, minimum temperature 22.9 °C accompanied by night RH 92% and day RH 64%.

Management

- Crop rotation with sugarcane and wheat is important for inoculum reduction of this soil pathogen.
- All infected plants must be uprooted and burnt along with field sanitation by removing all the weeds.
- Summer ploughing followed by once irrigation in between April to June and then again ploughing to reduce the activated soil inoculum.
- Green manuring in June and July followed by soil application of *Trichoderma* @5 kg/ha soon after ploughing of the sun hemp.
- Seedlings dip in *Trichoderma* @1% suspension for 10 minutes before transplanting.
- Drenching of *Trichoderma* @1% suspension after 15 days of transplanting and repeat at 15 days interval if required.
- Maintain proper drainage and aeration in the field.
- Balanced dose of fertilizers including micronutrients is effective for reducing the disease.

References:

- Bahadur A, Dutta P (2021) Diseases of vegetable crops and their management. (Eds. Bahadur and Dutta), Published by NIPA, New Delhi, ISBN: 9789390591091, pp360.
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Name of disease: SCLEROTINIA BLIGHT/SCLEROTINIA ROT

Geographical origin, distribution and crop losses: The pathogen, *Sclerotinia sclerotiorum* is widely distributed and attacks a wide variety of hosts. The disease has been reported on brinjal from Tarai region of Uttarakhand and Katrain area of Himachal Pradesh. More than 40 per cent of the plants were infected in Katrain area of Himachal Pradesh and reduced seed weight and germination of seeds.

Symptoms: Infection takes place on leaves, twigs, flowers and fruits. Water soaked lesions develop and the infected tissue macerated by the pathogen. Plants start dying from apical portion because primary infection



Fig.24: Symptoms of Rhizoctonia root rot in brinjal

always takes place on flowers leads to rotting of adjoining plant parts (**Fig. 25**). Soft, wet, water-soaked rotting of stem, leaves, fruits and flowers are observed followed by white fungal growth over it (**Fig 26**). Mycelium develops honeydew stage soon after colonization. Later on, after drying the entire portion of pith and fruits is converted in to compact mycelial mat followed by hard sclerotial body. These sclerotia become black after drying. Infection on lower portion of main stem is resulting complete wilting of brinjal plant. The pathogen is soil borne as well as concomitant seed mixture.



Fig.24: Symptoms of Rhizoctonia root rot in brinjal



Fig. 26: Symptoms of Sclerotinia blight on stem and leaves of brinjal

Causal organism: *Sclerotinia sclerotiorum*

Key Characteristics: Mycelium in culture as well as on host surface is hyaline, cottony, branched, septate hyphae (9.0 to 18.0 μm broad). Microconidia (1.5 to 3.5 μm) are produced on short lateral branches of the mycelium in chains. The pathogen produces sclerotia at the periphery of the colony, which initially are white and later turn black in colour, round to irregular in shape. The sclerotia also germinate by producing stalked apothecia, which ranges one to five per sclerotium under suitable humidity and light conditions. Apothecia are brown in colour and are round or lobate type. Ascii are cylindrical measuring 108-153 x 4.5-10 μm in size. Each ascus contains eight ascospores and is released in clouds. Ascospores are elliptical and varies from 7-16 x 3.6-10 μm in size.

Disease cycle and favorable conditions: Sclerotia germinate by forming apothecium which in turn forms ascii under humidity and light condition. Sclerotia are also capable of infecting the plants. After infection, the fungus produces oxalic acid, which kills the cells in advance. A fluffy mycelium appears on the host surface and cool and moist during scarcity of food for fungus weather the mycelium coagulates and starts forming sclerotia, which are initially white in colour, but later turns black. The fungus survives in soil in the form of sclerotia over a wide range of temperatures i.e. from 0 to 25°C with an optimum at 15 to 20°C. High moisture and low temperature conditions are necessary for infection. The fungus can tolerate wide pH range but is best adapted to an acidic substrate.

Management

- The following management practices may be followed against Sclerotinia rot. Seed must be free from sclerotia and seed infection. If sclerotia are observed in the seed lot, these should be removed with floatation. Seed borne mycelium can be inactivated by seed treatment with fungicide like iprodione + thiram. Seed treatment can also be carried out with the biocontrol agent like *Trichoderma harzianum* or *T. viride*.

- Soil borne inoculums in form of sclerotia should be deactivated with help of soil drenching fungicides. Sclerotia bearing plant parts should be destroyed and burnt immediately. Burying the sclerotia deep into the soil by ploughing at least for 30 weeks ensure destruction of them. The soil should be kept weed free as most of the common weeds are susceptible to this disease and harbor Sclerotinia fungi to build up to high levels in the soil.
- Crop debris and infected plant parts should be carefully cut along with some healthy part and collected from the field in morning. These should be burnt without shattering and falling of sclerotia in the field. Avoid excessively high plant populations and narrow row spacing.
- Crop rotation and no tillage is the most useful combination of treatments to reduce primary inoculums. Crop rotation with flooded rice is beneficial for reducing the soil inoculum.
- Destruction of sclerotia by antagonistic fungi, bacteria and soil amoeba is also reported. The fungal parasite of sclerotia, *Sporodesmium sclerotivorum* and *Coniothyrium minitans* have also the potential to keep the numbers of inoculums in lower level.
- Spraying of ziram, ferbam or systemic fungicides of benzimidazole group have proved effective in checking spread of the disease. Tetrasodium thiocarbamate and the dicarboximide fungicide such as procymidone, vinclozolin and iprodione are highly effective against *Sclerotinia*. Foliar spray of mancozeb @0.2% at flowering stage followed by spray of copper oxychloride 50%WP @0.25% in cool, cloudy and humid weather.
- There is a limitation to develop commercial plant resistance against Sclerotinia fungi. Disease avoidance is the most suitable technique. Reduction of the population of sclerotia surviving from one crop to the next is the essential to get rid of from this destructive disease.
- Summer ploughing in such a way that surface is buried deep.
- Green manuring in June and July followed by soil application of *Trichoderma* @5kg/ha soon after ploughing of the sun hemp.

References:

Bahadur A, Dutta P (2021) Diseases of vegetable crops and their management. (Eds. Bahadur and Dutta), Published by NIPA, New Delhi, ISBN: 9789390591091, pp360.

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Name of disease: COLLAR ROT

Geographical origin, distribution and crop losses: It has wide geographic diversity and commonly found in the tropics, subtropics and other warm temperate regions especially in the Southern United States, Central and South America, West Indies, Southern European countries bordering the Mediterranean, Africa, India, Japan, Philippines and Hawaii and in warmer region like Bangladesh. Collar rot may cause up to 30-50 % loss in fruit yield in eggplant.

Symptoms: It has wide host range covering almost all vegetable crops. The first symptom of disease is observed as soft tissue necrosis of bark of the stem near soil line. White, cotton like and silvery mycelium growth is clearly visible on the affected portion (Fig. 27). Fungal growth was also found just below the soil surface. Later,



Fig. 27: White cottony growth of *Sclerotium rolfsii* causing collar rot in brinjal

white to light brown mustard like sclerotia is observed on the same portion. Progressive dropping and yellowing or wilting of the entire plant is observed. Sometime plants collapse soon after infection. The disease is soil borne where pathogen survives in the form of sclerotia.

Causal organism: *Sclerotium rolfsii*

Key characteristics: *S. rolfsii* is a ubiquitous, necrotrophic soil fungi. It exhibited white cottony mycelial growth with regular shaped colony. Mycelial growth is fluffy to dense compact in the centre. Sclerotia formed by the fungus at the edges of the plates from 5 days after inoculation.

Disease cycle and favourable conditions: Mechanical spread of *S. rolfsii* sclerotia occurred by soil attached to shoes, hand tools, vehicle tires, or farm machinery or in splashing water. Long-distance spread of sclerotia occurs through plant material or soil during shipment. Under favorable weather conditions, sclerotia resume activity by either eruptive or hyphal germination in which white mycelium burst out of the sclerotial rind. An external food source is not required for this type of germination. Sclerotia can germinate eruptively only after being induced by dry conditions or volatile compounds. Sclerotia can germinate hyphally more than once. Growth of individual hyphae from sclerotia is in response to availability of exogenous nutrients.

Management

- Carefully select crop rotation with cereals, corn, sorghum and millets because the pathogen is having broad host range.
- Remove all the weeds particularly dicot from field.
- Avoid use of infested farmyard manure, soil, compost etc. in the field.
- Irrigate the soil in summer and plough the field to destroy germinating sclerotia.
- Green manuring followed by application of *Trichoderma* formulation@5kg/ha within a week of ploughing.
- Seedlings root dip in *Trichoderma* @1% for 10 minutes.
- Avoid dense planting and maintain proper aeration near collar region.

References:

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- Singh RS (2004) Plant Diseases. Oxford & IBH Publishing Co. Pvt. Ltd. New Delhi.

Name of disease: PSEUDOCERCOSPORA LEAF BLIGHT

Geographical origin, distribution and crop losses: This disease was first time observed during winter season in the month of January 2001 and it was first report from India.

Causal organism: *Pseudocercospora* spp.

Symptoms: Olivaceous black sooty growth of fungus was observed on the lower surface of the leaves (Fig 28). Symptoms are only observed on the leaves. Leaves defoliate soon after infection. No discoloration was observed on the corresponding upper side of the leaves. *Pseudocercospora* spp. was first time recorded on brinjal as a new host and may spread in future.



Fig. 28: Symptoms of Pseudocercospora leaf blight

Management

- Collect and burn the infected leaves.
- Carefully select exotic hybrids and varieties.
- Need based one or two sprays of zineb 75%WP @0.2% alternated with mancozeb 75%WP @0.25%

References:

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Name of disease: BACTERIAL WILT

Geographical origin, distribution and crop losses: Kelman (1953) was the first to review the literature and indicate the worldwide economic importance of bacterial wilt on many hosts. In India, the disease is known to occur in Punjab, Himachal Pradesh, Madhya Pradesh, Bihar, West Bengal, Assam, Odisha, Kerala, Karnataka, Tamil Nadu, Jammu and Kashmir, Uttarakhand and Jharkhand. Up to 90.6 per cent loss in tomato yield due to this disease has been reported in some badly affected fields in India.

Symptoms: The characteristic symptoms of the disease are drooping of lower leaves, wilting, stunting, and yellowing of the foliage followed by full-plant wilting (Fig. 29) and brown discoloration of vascular system. Milky white bacterial ooze is produced when cut ends of wilted plant are placed in water. Development of adventitious roots from the stem is considerably increased. Tendency of excessive adventitious root formation depends on the number of vascular bundles invaded. Adventitious roots develop just outside the invaded vascular bundle. Presence of nematode in the soil predisposes the infection. Sudden drooping of leaves without yellowing and rotting of the stem is observed if wet weather with high temperature prevails for considerable period. The bacteria multiply rapidly in the xylem vessel and finally block the translocation of water which causes wilting of the plant. All parenchyma cells near to the vessels with bacteria become necrotic. The pit membranes are often thicker with high electron density. After collapse of the plant the bacteria were found to be released into the soil.

Causal organism: *Ralstonia solanacearum*

Key Characteristics: The bacterium is gram negative, rod shaped, frequently occurs in pairs, motile with 1 to 4 polar flagella and measures 0.5-0.7 x 1.5-2.5 μm (Fig. 10). It is aerobic and catalase and oxidase-positive and forms nitrites from nitrates. It, like some other non-fluorescent pseudomonads, produces intracellular, refractile, sudsophilic inclusions consisting of polyhydroxy-butyric acid. Some strains produce a brown diffusible pigment on complex



Fig. 29: Symptoms of bacterial wilt of brinjal

media and a black pigment on autoclaved potato slants. The pathogen is negative for levan production and starch hydrolysis. It causes weak hydrolysis.

Disease cycle and favorable conditions: Once inside the host, the bacterium has an affinity for the vascular system where it multiplies rapidly filling the xylem vessels with bacterial cells and slime. Wilt occurs within 2-5 days after infection depending upon host susceptibility, temperature and virulence of the pathogen. As the host wilts, the pathogen invades parenchyma cells in the pith and cortex and pockets filled with masses of bacteria develop around the vascular bundles. Under favorable conditions the bacterium may move through the cortex and exudes on the surface of the stem. It is released into the soil from the roots of the infected plants and from decaying host material. Optimum temperature for the growth of the bacterium is 35-37°C. Disease does not develop at 15°C and most favorable temperature for rapid development of the disease takes place at 37°C. The disease mostly develops, when mean temperature is above 20°C.

Accumulation of oxidized phenolics and plant cell break down products are responsible for internal browning of the vascular tissues. Brown discoloration of the vascular bundles can be observed by splitting the stem and roots longitudinally.

Management:

The following management practices may be followed.

Selection of disease-free field: The selected field should have no previous history of bacterial wilt disease. The field in which susceptible solanaceous host had grown during the previous year should not be selected. Disease incidence can be successfully reduced with regular rotation with other non-host plants. However, short duration rotation (one year) with paddy doesn't make the field disease free.

Use of pathogen free seedlings: Nursery for raising brinjal seedling should not be in the fields with the history of bacterial wilt. Fumigation or soil solarization of soil 15 days prior to sowing is recommended.

Prevent the spread of the pathogen: Immediate removal of infected plants is strictly recommended. The infected portion of the field should be isolated immediately by preventing the water flow into and from the field. Frequency of irrigation should be reduced. Flood irrigation in the infected field should be avoided as it increases the disease incidence by two-fold. Movement of people/machinery should also be restricted.

Reduction of the pathogen load: The following methods are effective in reducing the pathogen load in the infected field. (a) Crop rotation for 3-4 years with paddy and other non-host crops (b) Flooding the field for 1-3 weeks before planting (c) the incidence of nematode can be reduced by growing marigold (*Tagetes* spp.) in rotation or as intercrop (d) enhance the activity of beneficial microflora by application of organic manures like FYM/ vermicompost every year.

Biological control:

- Nursery application: Seed treatment with talc based consortial formulation of antagonistic biocontrol agent viz. *Pseudomonas fluorescens* and *Trichoderma harzianum* (10 g/100 g of seeds).
- Root dip treatment: Mixing of 1 kg of the consortial bioformulation with 1 L of starch solution (rice gruel) and keep the root of the seedling for an hour before transplanting. One kg of the formulation is enough for root dip treatment of 1000 numbers of seedlings.
- Soil application: one kg of the consortia of *Pseudomonas fluorescens* and *Trichoderma harzianum* are mixed with 10 kg of dry cow dung or vermicompost and apply @ 100 g per plant.

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Name of disease: LITTLE LEAF & PHYLLODY

Geographical origin, distribution and crop losses: This disease was first reported from India in 1938. Since then, it has also been reported from Sri Lanka and Bangladesh. In India, this disease has been recorded from almost all states wherever brinjal is grown. The incidence of the disease when assessed in Warangal district of Andhra Pradesh, it was reported to be less than 8 per cent the reduction in the primary productivity of little leaf infected plants is reported to be 64.2 per cent.

Symptoms: The major symptoms associated with the disease are little leaf, leaf yellowing witches' broom, phyllody, proliferation of auxiliary buds, stunted plant growth, shortening of internodes and reduced abnormal and malformed fruits (Fig. 30). The flower bud become upright and produces phylloid flowers. Setting of fruits appear to be highly reduced in plants which experience infection in late stage but, contrary to it, the plants which get infected during early stage of their growth completely fail to develop any fruit. There is 100% loss caused by the disease.

Causal organism: The disease is caused by Clover proliferation phytoplasma related strains (16Sr VI-A, B, D subgroups), *Ca. P australasia* (16SrII group); *Ca. P. fraxnii* (16SrVIII-A subgroup) (Rao and Kumar, 2017).



Fig. 30. Phytoplasma associated symptoms in brinjal: Little leaf, phyllody and shoot proliferation

Key Characteristics: The disease is caused by a Phytoplasma. Phytoplasma bodies (230-770 nm) are found associated with the phloem cells of roots of the eggplant affected with little leaf. Many alternate hosts and natural leafhopper vectors are known as natural reservoirs for these phytoplasma strains associated with brinjal phyllody and little leaf in India (Rao 2021). Each phytoplasma body contained ribosomes and strands of nuclear material surrounded by 16.5 µm wide triple layer unit membrane. Electron micrographs revealed pleomorphic phytoplasma bodies in the sieve elements of the affected plants. The disease is transmitted from one plant to other and is spread in the field to field by leaf hopper vector *Hishimonas phycitis*. The disease is not sap transmissible. Graft transmission and dodder transmission is found successful.

Disease cycle and favorable conditions: In nature, the phytoplasma perennates on several weed hosts like *Datura* spp. and *Vinca rosea* and several others. The vector *Hishimonus phycitis* transmits the disease from weed hosts to healthy plants. Early sown crops (June sown) escape the disease compared to July and August sown crops due to differences in vector populations. Continuous cropping of the susceptible varieties increased disease incidence, as did the presence of weeds, sweet potato or tomato crops in the vicinity, which helps to increase in vector populations.

Management

- Periodical spray of systemic insecticides such as Thiamethoxam 25 WG @150 g/ha or imidacloprid 17.8 SL @150 ml per hectare is necessary to control leaf hopper vector.
- Uproot the severely infected plants and burn just before starting of spray schedule.
- Treatment of seedlings with (Streptomycin Sulphate + Tetracyclin hydrochloride @ 150 ppm) for 20-30 minutes + installation of yellow sticky traps @ 15/ha after 20 days of transplanting + destruction of the infected plants + need based foliar application of Streptomycin Sulphate + Tetracyclin hydrochloride @ 150 ppm and the insecticide Imidacloprid @ 0.3 ml per liter.
- Temporary remission of symptoms by tetracycline (Anjaneyulu and Ramakrishnan, 1973; Raychaudhuri et al., 1970).
- Severity of the phytoplasma disease could be reduced by roughing symptomatic plants and spraying insecticides
- Selection and screening of brinjal resistant genotypes resistant to phytoplasma infection
- Wild relatives of brinjal (*Solanum integrifolium* and *S. gilo*) were resistant to the little leaf disease
- Eradication of weeds that harbor phytoplasma and its insect vector, along with spraying insecticides may be significant in minimizing the eggplant phytoplasma disease incidence in the fields.

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Chilli

Introduction

Chilli peppers are varieties of the berry-fruit of plants from the genus *Capsicum*, which are members of the nightshade family Solanaceous, cultivated for their pungency. There are five domesticated species of chilli peppers and *Capsicum annuum* includes many common varieties such as bell peppers, wax, cayenne, jalapeños. Capsaicin and related compounds known as capsaicinoids are the substances giving chille peppers their intensity when ingested or applied topically. Other varieties of capsicum include bell peppers (sweet peppers), but while *chilli peppers* are pungent, bell peppers are not. Chilli is a very remunerative, widely grown indispensable spice as well as cash crop to the world food industry and is cultivated in almost all states of the country. Chilli pepper pods are, technically, berries. When used fresh, they are most often prepared and eaten like a vegetable. Whole pods can be dried and then crushed or ground into chilli powder that is used as a spice or seasoning. Chillies can be dried to prolong their shelf life. Chilli peppers can also be preserved by brining, immersing the pods in oil, or by pickling. India is the major and highly erratic producer, consumer and exporter of chilli. About 80% of Indian chilli is consumed domestically and the remaining is exported to other countries. However, Indian chilli consignments for export face twin challenges of pesticide residues and aflatoxin contamination. Chilli crops suffer with various fungal diseases such as damping off (*Pythium* sp.), Anthracnose/Fruit rot/Die back (*Colletotrichum capsici* and *Colletotrichum gloeosporioides*), Fusarial wilt (*Fusarium solani* and *F. oxysporum*), Rhizoctonia root rot (*Rhizoctonia botaticola*), Cercospora leaf spot (*Cercospora capsici*) and Alternaria leaf spot (*Alternaria solani*), bacterial diseases like bacterial wilt (*Ralstonia solanacearum*) and leaf spot (*Xanthomonas* sp.) and viral diseases such as such as mosaic, tospo, illar and leaf curl. In addition, Aflatoxin contamination (*Aspergillus flavus* and *A. parasiticus*) and fruit rots caused by *Colletotrichum* sp., *Alternaria* sp., *Fusarium* sp. and *Rhizopus* have been reported as post harvest diseases on chilli.

Name of disease: DAMPING OFF

Geographical origin, distribution and crop losses: Damping off of Chilli cause 62% seedling mortality and 90 per cent of plant death both at pre or post-emergence stage in the nursery, fields and greenhouse. It can cause serious loss under field and greenhouse condition affecting newly emerging seedlings and infecting the hosts by rapidly infesting germinating seeds, therefore creating obstruction in management. This disease is considered as an important limiting factor in successful cultivation of crop plants throughout the world.

Symptoms: Damping-off of seedlings occurs in two stages i.e., pre- and post-emergence stage. Post -emergence damping off is more conspicuous than pre-emergence phase, which often go undetected except resulting into poor crop stand. In pre-emergence phase, young seedlings disintegrate, get killed before reaching soil surface. The radicle and plumule rot before hypocotyl break seed coat, leading to poor field emergence or seed

germination. In post-emergence phase, disease develops after seedlings have emerged out of soil, causing infection in stems before lignification. The disease is characterized by lesion formation at collar region, appearance of brown water-soaked areas and softening of tissues. Infected stems become hard, constricted at base, thin i.e., wire stem symptoms, shrivel and collapse, ultimately toppling over giving patchy appearances both in nursery and main fields. In nursery and field conditions, damping-off infection usually radiates from initial infection points causing large spots or areas killing large number of seedlings. Generally, cotyledons and leaves wilt before seedlings are prostrated, but in some cases, seedlings remain green and turgid until they collapse.

Causal organism: *Pythium aphanidermatum* (Edson) Fitz. Several soil inhabiting fungi predominantly *Pythium* spp. viz., *P. aphanidermatum*, *P. debaryanum*, *P. ultimum* cause seed rot and damping off of chilli seedlings.

Key characteristics: The pathogens associated with this disease are either seed or soil borne or both. Species of *Pythium*, *Phytophthora*, *Fusarium*, *Sclerotium* attack the plants when they are in juvenile stage. In chilli crop two species of *Pythium* have been reported. i.e *Pythium ultimum* Trow. Fitz. and *Pythium aphanidermatum* (Edson) are reported to cause damping off. Among them *P. aphanidermatum* is more in habitats like hydroponic culture and low-lying areas, while *P. ultimum* generally occurs relatively in dry areas. They infect many hosts unlike *Phytophthora* which is host specific. Soil moisture near saturation stimulates mycelial growth as well as asexual reproduction. Propagules capable of causing infection include vegetative mycelium, germinating sporangia, zoospores and oospores. Some propagules, particularly zoospores may meet chilli plants by chemotaxis. Infective propagules are capable of direct penetration, but wounds enhance penetration and infection.

Disease cycle and favourable conditions: Damping off of chili is soil borne in nature and practice of monoculture together make the crop susceptible to disease. The cultivars grown are not resistant to the pathogen which also helps to spread the disease. The disease becomes severe at high temperature and humidity. Seedlings grown in the atmosphere having high density, high humidity and high temperature favours the disease development. The adult plants are more resistant to damping off. Factors which favors damping off in chilli includes, moist soil poor drainage with 90-100% relative humidity and soil temperature around 20°C. The disease is further aggravated high density of seedlings and poor drainage, low lying areas, clayey soils, flat beds with low content of sand and continuous cultivation in the same field.

Management

Cultural:

- Selection the site prepared by deep summer ploughing
- Adopt sanitary precautions such as collection, destruction of crop debris and slow burn of farm waste such as paddy husk, grasses etc.
- Sow seeds at low seed rate @ 3 g/ha on raised nursery seed beds of light soil up to 15 cm high with proper drainage provided through channels
- Avoid overcrowding, maintain optimum spacing between seedlings and regulate irrigations to maintain optimum moisture on bed surface
- Compost of paper mill sludge + plant waste + manure and *Brassica* seed pomace (cake) + *Pseudomonas boreopolis* are found to suppress *Pythium* sp. damping-off disease

Biological:

- Seed coating with *Trichoderma harzianum*, *T. hamatum*, *Penicillium oxalicum*, *Burkholderia cepacia* and *Pseudomonas flourescens* @ 5g/Kg seed found effective biological control method against damping-off
- *P. oligandrum* as mycoparasite of *P. aphanidermatum*, *P. ultimum*, *P. graminicola* and other pathogenic *Pythium* spp.
- Garlic extract @ 10% has fungicidal properties against *P. aphanidermatum*

Chemical:

- Use seed protectants such as agrosan or ceresin or thiram or captan @ 2-3 g/Kg and systemic fungicides such as Ridomil (Metalaxyl) @ 0.2% as seed dressing
- Soil drenching of nursery beds @ two to three times with 1 % Bordeaux mixture or Copper oxychloride (COC) @ 0.3% or Ridomil (Metalaxyl) @ 0.2% starting from three weeks after sowing at 4 days interval

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Name of disease: DIEBACK AND ANTHRACNOSE

Geographical origin, distribution and crop losses: Severely infected by anthracnose which may cause yield losses of up to 50% (Pakdeevaporn et al., 2005). In India, a yield loss of 10–54% was reported due to the anthracnose disease (Ramachandran and Rathnamma, 2006). In other parts of the world as well, like a significant amount of 20–80% loss was recorded from Vietnam and about 10% from Korea. The loss is high owing to the post and pre-harvest involvement of the pathogen causing a loss of 10–80% of the marketable yield of chilli fruits (Than et al., 2008).



Fig. 31. Symptoms of Anthracnose lesions on infected twigs



Fig. 32: Symptoms of anthracnose on chili fruits

Symptoms: The disease is causing 20% yield loss due to dieback and 10% as fruit rotting every year. Symptoms initiated on tender twigs from the apical top of the plant as necrosis and withering (Fig. 31). It progresses from tip to downward. *Choanephora capsici* rotted leaves and twigs clearly show black sporangial structure over it. The twigs are water soaked, brown and dieback as the infection spreads downward. Later on, infected twigs developed several minute acervuli. Symptoms on red fruits are more commonly observed than green fruits. Small, irregular, sunken, light brown lesions were observed on mature fruit (Fig. 32).

Most oftenly they are coalesced and become papery on green fruits. Acervuli formation started from the center of lesion and moved toward periphery on red fruits. Sometime arrangement of acervuli was in concentric ring fashion. Apparently, fruits looking healthy during harvest may show the symptoms of disease during drying. Maximum infection was observed in first fruiting. Fruit rot extends to the seed cavity making it internally seed borne pathogen.

Causal organism: Dieback is caused by *Choanephora capsici* and *Colletotrichum capsici* both while Anthracnose is only caused by *Colletotrichum capsici* and *C. gloeosporioides*.

Key characteristics: *Colletotrichum* is an asexual genus belonging to phylum Ascomycete and Coelomycetes class of Fungi imperfecti. Despite significant developments in studies related to this plant-patho system, the taxonomic position of the pathogen remains unclear.

Disease cycle and favorable conditions: Most *Colletotrichum* spp. are seed borne and may overwinter in soil or on infected crop debris. Conidial dispersal takes place by rain splash and the transmission of ascospores takes place through the air. Seeds and soil harbor *Colletotrichum* conidia and overwinter as micro sclerotia in soil. Weather variables such as optimum temperature of around 27 °C, leaf wetness, relative humidity of 80 per cent and above with soil pH of 5-6 virulent pathogen and susceptible host cultivar favors disease proliferation.

Management:

- Disease free seeds should be collected from healthy fruits.
- Screening of diseased fruits must be done after drying of the fruits.
- Seeds should be treated with carbendazim @0.25% during sowing.
- Seedling should be sprayed by carbendazim @0.1% before transplanting.
- Cut the rotting twigs along with healthy part and burn it.
- Alternate foliar spray of azoxystrobin 11%+tebuconazole 18.3% w/w SC @0.1% and myclobutanil 10% WP @0.04%.
- Avoid apical injury during transplanting and at flowering stage.

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Name of disease: BACTERIAL LEAF SPOT

Geographical origin, distribution and crop losses: The bacterial leaf spot of squash was described in 1926, which is distinct from angular leaf spot and causes maximum damage to cucumber. It was reported from India in 1931 on cucumber. The disease was reported on squash, pumpkin and water melon.

Symptoms: The disease is common from nursery stage to vegetative stage of plant growth. Symptoms may appear any time up to first flowering. Very small, black, circular spot surrounded by yellow halo mostly observed in leaves (Fig. 33). Affected lower leaves become yellow and fall down. Bacterial spots remain green and appear as spot bending symptoms on completely yellow leaves.

Causal organism: The disease is caused by *Xanthomonas cucurbitae* (ex Bryan 1926) Vauterin et al. (1995).

Key characteristics: On beef agar, the colonies are mustard-yellow. Yellow-pigmented *Xanthomonas* like bacterial colonies were isolated on KC semi selective medium (yeast extract 7 g, peptone 7 g, glucose 7 g, agar 18 g, distilled water 1,000 ml, propiconazole 20 g ml, cephalexin 40 mg liter⁻¹, and kasugamycin 20 mg liter⁻¹). The bacterium is a rod shaped, motile with a single polar flagellum, gram-negative, O+ and F- in the oxidative and fermentative test, and oxidase negative. The bacterium hydrolyzed starch and esculin, but did not hydrolyze nitrate, and grew on YDC agar at 33°C.



Fig. 33. Bacterial leaf spot

Disease cycle and favorable conditions: The bacterium is seed borne and persists in infected crop residue. The infection of seed occurs internally in the seed coat. The cotyledons are infected after germination of the seed. The disease is transmitted by seeds as well as rain splash and the movement of people and machinery. Symptom expression usually occurs during periods of high temperature generally following rain or overhead irrigation. The disease is favored by warm temperatures and high humidity or wet conditions caused by frequent rains or overhead irrigation. Optimum temperature range is from 24-30°C and maximum 35°C for the growth of the bacterium.

Management

- Summer ploughing to desiccate the bacteria and host.
- Soil solarization in nursery bed to avoid seedling infection.
- Rotation of nursery seedbed and main field.
- Seed should be collected from disease free plants.
- Foliar spray of copper oxychloride @0.25% in afternoon.

References:

- Singh D, Chowdappa P, Sharma P (2014). Diseases of Vegetable Crops: Diagnosis and Management, Indian Phytopathological Society, New Delhi- India, pp. 746
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Name of disease: WHITE ROT

Geographical origin, distribution and crop losses: Among the fungal diseases Sclerotinia rot caused by *Sclerotinia sclerotiorum* is one of the most devastating soil borne pathogen, which is threatening the

production of chilli crop. The pathogen attacks nearly all kinds of succulent plants including flowers, shrubs, weeds and vegetables including chilli.

Symptoms: The disease is observed in December to January during low temperature, cloudy weather and high atmospheric humidity accompanied by soil moisture. The disease cause infection at all stages of the susceptible crops viz. French bean, chilli, potato, brinjal etc. The primary symptom of the disease is the presence of wilted plants in a field of healthy-looking plants. Infection occurs mainly on stems and branches. As a result of infection, under severe conditions, the portion of plant beyond the infection wilts. Soft, wet, water-soaked rotting is observed on stem followed by white fungal growth over it. Infected stem turns white after drying (Fig. 34 & 35). This mycelium develops honeydew stage soon after colonization. The pith region is filled with brown to black coloured sclerotia. In case of infection in the fruits, rotting of flesh occurs with numerous sclerotia.



Fig. 34. White rot symptoms on chilli plant



Fig. 35. Infection of *Sclerotinia* on stem of chilli plant

Causal organism: White rot of chilli is caused by soil-borne fungus *Sclerotinia sclerotiorum*.

Key characteristics: The pathogen is capable of reproducing both sexually and asexually. The sclerotia are of two to three thick layers of hyphal aggregates, ring, medulla and cortex. The outer ring is composed of melanin incorporating the black colour.

Disease cycle and favourable conditions: The ascospores of *S. sclerotiorum* only infect the flower of susceptible hosts and begin to invade the host's tissues via mycelium, causing infection. *S. sclerotiorum* can invade nearly all tissue types including stems, foliage, flowers, fruits, and roots. Upon infection white, fluffy mycelium will grow on the surface of the infected tissues. *S. sclerotiorum* once again will produce sclerotia at the end of the growing season. The sclerotia will then remain on the surface of soil, until the next season. The pathogen proliferates well in moist and cool environments. Under moist field conditions, *S. sclerotiorum* is capable invading colonizing nearly all the plant's tissues primary host with mycelium. Optimal temperatures for growth range from 15 to 21 °C.

Management

- Cut the infected plant parts along with some healthy portion in morning and carefully collect in polythene to avoid falling of sclerotia in the field. Burn all these materials away from field.
- Use of disease-free planting material.
- Collection and destruction of diseased parts and portions of plants.
- Crop rotation with non-host crops like maize and rice.
- Deep summer ploughing should be done.
- Seed treatment with 5 g of *Trichoderma viride* formulation per kg of seeds will help to reduce the disease.
- Foliar spray of carbendazim @0.1% at flowering stage followed by spray of mancozeb @0.25%.

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Name of disease: LEAF BLIGHT

Geographical origin, distribution and crop losses: Leaf blight of chilli caused by *Alternaria alternata* is one of the most destructive disease in India which are seed borne and reduce the seed germination and yield loss up to 30-60 per cent. Prevalence of cercospora leaf spot disease of bell pepper caused by *Cercospora capsici* in seven growing locations of Kashmir, India, at fruit set (25th July to 5th August) and fruits development (10-20th September) stage. Li et al. (2011) reported that 70-80 per cent chilli fields are affected with *Alternaria* spp. in Shouguang district.

Symptoms: Symptoms of Cercospora leaf blight are observed as circular to oblong spots with light gray to white gray centers and dark brown margins (Fig. 36). Small spots coalesce to each other in severe case and leads to defoliation. Alternaria spots are dark brown, irregular and covered with a grayish brown to black spores of the pathogens. In moist condition, sporulation of both pathogens was observed on the spots.

Causal organism: Two pathogens are associated with leaf blight disease of chilli are viz. *Alternaria alternata* and *Cercospora capsici*.



Fig.36: Symptoms of leaf blight on chilli

Key characteristics: The genus Cercospora poses a major challenge for systematics because the phylogenetic relationships of many of its members still are unclear. Swamy et al. (2011) obtained 15 isolates and identified them as '*Cercospora capsici*' and are markedly similar to the reference strain CPC 12307 of *C. capsici* which is the first report from India. The information provided by these studies gives more useful tips for assessing the Cercospora taxonomy.

Disease cycle and favorable conditions: Pathogen grow luxuriantly at less than 28°C temperature, 92% relative humidity and pH 5-6. Below 90% relative humidity, the disease does not develop. The fungus survives in plant debris, primary infection coming from air-borne spores derived from it. The disease is more severe in wet weather than in dry weather and becomes destructive in high relative humidity.

Management

- One foliar spray of zineb 75%WP @0.2% alternated after 10 days by azoxystrobin 8.3% + mancozeb 66.7% WG @0.2%.
- Selections of disease free and certified seeds to check the primary infection.
- Foliar spray of micronutrient @0.2% to maintain crop vigor at 10-12 days interval.
- Field sanitation by burning of infected crop debris followed by summer ploughing.

References:

Sharma P, Jambulkar PP, Raja M (2021) Management of the diseases of vegetable crops through fungal biopesticides. In: Biopesticides in organic farming. CRC Press, Florida, USA, pp. 91-96.

Bahadur A, Dutta P (2021) Diseases of vegetable crops and their management. (Eds. Bahadur and Dutta), Published by NIPA, New Delhi, ISBN: 9789390591091, pp360.

Name of disease: LEAF CURL COMPLEX

Geographical origin, distribution and crop losses: Etiology of Chilli leaf curl was established during the 1960s in India. Chilli leaf curl epidemic occurred during 2004 in Jodhpur, the main chilli-growing area in Rajasthan, India and then Senanayake et al. (2007) have reported first time chilli leaf curl virus on chilli crop from Jodhpur (Rajasthan). Recently identification of tomato leaf curl virus (ToLCV) strain causing leaf curl disease in Tomato and Chilli in Maharashtra reported by Chavan et al. (2013), results in 90-100% yield loss.

Symptoms: Reduction in size of leaves, shortening of veins, puckering, mottling of the leaves, stunting and bushy appearance of the plant are some of the symptoms of viral infection (Fig. 37). Conspicuous outgrowth and prominent veins are visible on lower side of the leaves. Downward rolling, narrowing and shortening of leaves in mite infestation (Fig 38).



Fig. 37: Symptoms of chilli leaf curl

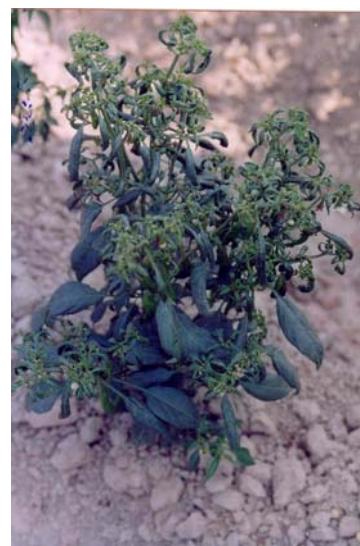


Fig. 38: Downward rolling of leaves due to mites infestation

Causal organism: Chilli leaf curl complex is mainly due to CMV, TMV, leaf curl Begomo virus as well as mite and thrips. Chilli leaf curl virus transmitted by insect vector of family Aleyrodidae and order Hemiptera, the whitefly *Bemisia tabaci*.

Key characteristics: Genetically chilli leaf curl virus is a ssDNA virus of family Geminiviridae and genus Begomovirus. Preferred scientific name of virus is Chilli leaf curl India virus.

Disease cycle and favorable conditions: Temperature in the range of 15–35°C plays an important role in epidemic as both vector population and virus transmission are influenced by temperature.

Management

- Root dipping of the seedlings in imidacloprid solution @0.4 ml per liter of water for one hour during transplanting.
- Nursery should be grown under 40 mesh nylon net to check the vector infestation.
- Barrier crop of taller non-host crop like maize, bajra, sorghum.
- Collect healthy seeds from disease free plants.
- Alternate spray of wettable sulphur 80% WP @0.3% and chlorfenapyr 10.00% SC @ 0.15% and spiromesifen 22.90% SC @0.05% at 10 days interval.
- Avoid mechanical injury while intercultural operations.
- Initial rouging of infected plants soon after infection and burn it.

References:

Chavan AK, Choudhary RS, Narwade R, Haribhau AR (2013) Identification of Tomato Leaf Curl Virus (ToLCV) Strain Causing ToLCV in Tomato and Chilli. International Journal of Science and Research (IJSR) 4(8): 434438.

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CHILLI LITTLE LEAF & YELLOWING

The phytoplasma disease cause little leaf and leaf chlorosis in *Capsicum annuum* L. (Chilli). The entire affected plants become stunted with leaf chlorosis and yellowing with little leaves (Fig. 39). The symptoms look like caused by a begomovirus. Sometimes mixed infection of phytoplasma and begomovirus also has been recorded in India. The disease has been identified from Delhi, Tripura and Baharaich (Uttar Pradesh). The causal organism is aster yellows group (16SrI-J subgroup) and peanut witches broom group (16SrVI-D subgroup) (Rao et al. 2017).

Disease cycle and Epidemiology: by leafhoppers and alternate hosts. Management is suggested by spraying insecticides and roughing the symptomatic plants at early stage of infection.



Fig 39: Little leaf and leaf chlorosis symptoms in chilli caused by phytoplasmas

Okra

Introduction:

Okra (*Abelmoschus esculentus* L.) is one of the important vegetable crops belongs to the family Malvaceae and grown exclusively throughout the tropical, subtropical and warm areas of temperate zone of the word. Young fruits are consumed fresh or cooked. Its fruits are good source of vitamins A, B, C and protein, carbohydrate, fats, minerals iron and iodine. Okra seeds contain 26% protein and 18.1 % oil. The mature pods and stems of okra containing crude fibers are used for clearing sugarcane juice for making jaggery (gur) and in paper industry. Okra has a good potential as a foreign exchanger crop in India and accounts for 60% of the export of fresh vegetables. India ranks first in the world with 5,784.0 thousand tonnes (72% of the total world production) of ladyfinger/okra. In India, okra is grown on 376.1 thousand hac. Producing 3684.0 thousand tones with an average yield of 9.8 tones per hectares. West Bengal is the leading okra producing state which has production of around 718.9 thousand tons followed by Bihar, Andhra Pradesh and Karnataka. During cultivation the crop is severely affected by various diseases of fungal, bacterial and viral nature, which not only reduce the quantity but also affect the quality of the fruits and these diseases are damping off (*Pythium* sp., *Rhizoctonia* sp.), Fusarium wilt (*Fusarium oxysporum* f. sp. *vasinfectum*), powdery mildew (*Erysiphe cichoracearum*), Cercospora leaf spot (*Cercospora abelmoschi* and *C. malayensis*), Yellow vein mosaic virus (YVMV) and enation leaf curl disease

Name of disease: LEAF BLIGHT

Geographical origin, distribution and crop losses: The disease has been reported from many countries like Mexico, Puerto, Rico, India, China, Uganda and Egypt. In India the disease has been reported almost all states wherever this crop is grown. Losses from this disease vary with disease intensity and sometimes the infection is so severe that the leaves drop down prematurely. Leaf spot and blight caused by *Cercospora* species in okra are very important particularly in moderate temperatures and high humidity conditions.

Symptoms: The fungus causes no definite leaf spot but grow as sooty to dark olivaceous, ectoparasite covering entire leaf lamina (Fig. 40). Disease initiated as small isolated fungal growth on lower side of leaf but very soon it spreads on both sides. Pathogen reduces severely the photosynthesis area. Infected leaves fall very quickly after drooping and rolling. It causes severe loss in seed production crop as well as the late sown crop grown for green fruits. Sometime light brown lesions are observed after washing out of spores from leaves due to rainfall.



Fig. 40: Symptoms of leaf blight in okra

Causal organism: The disease is caused by *Cercospora abelmoschi* and *C. hibisina*

Key characteristics: The disease is caused by the three species of the *Cercospora* like *C. abelmoschi* ELL. & Ev., *C. hibicina* ELL. & Ev. and *C. malayensis* Stevens. These species differ in the size of their conidiophores and conidia. In *C. abelmoschi*, the conidiophores are long, brown and bear pale olivaceous, slightly tapered conidia. The conidiophores of *C. hibisina* are extremely long sometimes up to 1000 µm in size, narrow and bear conidia that are sometimes hyaline and appreciably narrower than those of *C. abelmoschi*. The conidiophores of *C. malayensis* are borne in clusters of 5-20 and bear conidia, which are colorless, narrow, long and tapering from the blunt base to the sharp tip.

Disease cycle and favorable conditions: *Cercospora* originates from soil-borne inoculum and previously infected plant material. It is spread during the season by conidia that dislodge from short white spore clumps on the surface of lesions. These fungi perennate in the infected plant debris and on the wild hosts. The three species cause infection in moderate temperatures (25–29°C) and high humidity. The disease (*C. abelmoschi*) was more serious, when the mean minimum and maximum temperatures were 17.9 and 25°C respectively, along with 80 per cent RH and heavy rainfall (330 mm).

Management

- Foliar spray of hexaconazole 2%SC @0.05% twice at 10 days interval on disease initiation.
- Spray should be started immediately after disease initiation; delay in spray schedule will not give proper control due to very fast secondary infection rate.
- Okra sowing should be completed latest by first week of July.
- Collect the defoliated leaves and burnt in field itself to reduce the inoculum at source.

References:

- Bahadur A, Dutta P (2021) Diseases of vegetable crops and their management. (Eds. Bahadur and Dutta), Published by NIPA, New Delhi, ISBN: 9789390591091, pp360.
- Sharma P (2007) Vegetables: Disease, Diagnosis and Biomanagement. Aavishkar Publishers Distributors, Jaipur-India
- Pandey PK, Pandey KK (2002) Emerging strategies to control fungal diseases in vegetables. In Applied Mycology and Biotechnology (Vol. 2, pp. 197-217). Elsevier.
- Pandey KK, Nagendran K, Tripathi AN, Manjunath M, Rai AB & Singh, B (2016) Integrated disease management in vegetable crops. Indian Horticulture, 61(1).

Name of disease: YELLOW VEIN MOSAIC (YVMV)

Geographical origin, distribution and crop losses: The YVMD was first reported in 1924 from India. The losses from this disease range from 49.3 to 94 per cent depending on the stage of crop growth at which the infection occurs and it varies from location to location and season to season.

Symptoms: The disease is very severe in *kharif* season. The viral disease is caused by yellow vein mosaic virus (Gemini virus) and transmitted by whitefly. Early infection causes 20-30% yield loss. Characteristics symptoms appeared as prominent yellow vein and veinlets having green tissue interveinal area (Fig. 41). Severely infected leaves some time become completely golden yellow. Fruits develop green to yellow colour and become hard in early stage of growth.



Fig. 41: Symptoms of okra yellow vein mosaic

Causal organism: The virus responsible for the disease is yellow vein mosaic virus (YVMV).

Key Characteristics: It belongs to the begomovirus group. The size of the virus is 18 x 30nm and has been reported to have close relationship with Indian Cassava mosaic begomovirus (ICMV) in ELISA tests using polyclonal antiserum. Virus particles are spherical, isometric measuring 28-30nm in diameter along with X-bodies as observed under electron microscope

Disease cycle and favorable conditions: During rainy season, the temperature and relative humidity might have been high enough to support disease development. Following this, in late rainy season, a fall in temperature might lead to a decline in vector population that could reflect in a reduced expression of disease.

Management

- Sowing in last week of June to first week of July.
- Removal of malvaceous hosts around the field.
- Twice rouging of infected plants at initial stage of plant growth.
- Periodical foliar sprays of imidacloprid 17.8% SL@0.4 ml per liter of water alternated with thiamethoxam 25.0% WG @0.3 g/liter of water at 10 days interval up to flower setting.

References:

Singh RS (2004) Plant Diseases. Oxford & IBH Publishing Co. Pvt. Ltd. New Delhi.

Jambhulkar PP, Singh V, Babu SR, Yadav RK (2013) Insecticides and Bioproducts against Whitefly Population and Incidence of Yellow vein mosaic virus in Okra. Indian J Plant Protect 41(3): 253-256.

Name of disease: ENATION LEAF CURL (ELCV)

Geographical origin, distribution and crop losses: In India, ELCD was first reported from Bangalore (Karnataka) during the early 1980s; causes yield loss up to 80–90%.

Symptoms: The disease is second most destructive viral disease of okra after YVMV in kharif season. The disease is caused by Okra enation leaf curl virus (Gemini virus) and transmitted by whitefly. The plant becomes unable to bear its weight after viral infection. The stem and leaf petiole are bent. (Fig. 42). Apical portion of plant developed into full of small, curl and aggregated leaves. Floral part does not set the fruit. Fruit if sets then seeds are unfertile and aborted. Characteristically small, green, outgrowth is observed on vein and veinlets of the lower side of leaves.



Fig. 42: Symptoms of Okra enation leaf curl

Causal organism: ELCD are caused by viruses of genus *Begomovirus* (family *Geminiviridae*).

Key characteristics: The geminiviruses are plant infecting viruses characterized by their unique geminate particle morphology and circular single-stranded (ss) DNA genomes that are transmitted by the whitefly *Bemisia tabaci* and infect dicotyledonous plants.

Disease cycle and favorable conditions: Hot weather with little or no rainfall was conducive for virus disease development and also for the multiplication of *Bemisia tabaci*, vector of ELCD.

Low rainfall caused significant outbreak in whitefly populations and dense population developed only when both humidity and temperature were high.

Management

- Wice rouging of infected plants at initial stage of plant growth.
- Periodical foliar sprays of imidacloprid 17.8% SL @ 0.4 ml per liter of water alternated with thiamethoxam 25.0% WG @ 0.3 g/liter of water at 10 days interval up to flower setting.

Name of disease: ROOT KNOT NEMATODE

Geographical origin, distribution and crop losses: Root knot nematode is ubiquitous and occur throughout the world. In India, fourteen species of root knot nematodes are recorded. Among them, *M. incognita* and *M. javanica* are widely distributed in different parts of the country. In addition to direct injury and migration, *Meloidogyne* acts as predisposing and facilitating agent for the entry of soil borne fungal and bacterial pathogens.

Symptoms: This is a severe problem of most of the vegetable crops in tropical and subtropical region. It has wide host range. Symptoms appear as stunting of the plants, yellowish green of the plant, sometime drooping of the leaves. Plant shows hunger sign. Clear symptoms are observed after uprooting of the plant where roots are full of knots. Main and lateral root bears spherical to elongated galls of variable size (Fig. 43). *Meloidogyne incognita* is dominant in India.

Causal organism: *Meloidogyne incognita* and *M. javanica*

Key characteristics: Female lays 400-500 eggs into gelatinous matrix generally protrude out of the host or some time embedded in it. Gelatinous matrix is glycoprotein complex produced from female rectal glands; act as protective agent against environmental extremes and predation.



Fig. 43: Root galls due to nematode infection in Okra

Management

- Apply neem cake 25g/ha before transplanting.
- Crop rotation with cereals and low land paddy for at least 2 years.
- Summer ploughing for desiccation of nematode.
- Sowing of marigold is effective for reduction of its population from soil.

Pea and Beans

Introduction

Pea (*Pisum sativum* L.) and different beans like French bean (*Phaseolus vulgaris* L.), lima bean (*P. lunatus* L.), scarlet bean (*P. multiflorus* Wild.), tepary bean (*Mucuna* sp.), field bean or lablab (*Dolichos lablab*), sword bean (*Canavalia ensiformis* DC.) and cluster bean (*Cyamopsis tetragonoloba* (L.) DC.) are the important leguminous crops which are grown as pulse crops and their green pods are used as vegetables. Peas are a good source of vitamins C and E, zinc, and other antioxidants that strengthen your immune system. Other nutrients, such as vitamins A and B and coumestrol, help reduce inflammation and lower our risk of chronic conditions, including diabetes, heart disease, and arthritis. In case of beans, they are unique among plant foods due to their high protein content. For this reason, they're considered an important protein source for vegetarians. Beans also contain decent amounts of zinc, copper, manganese, selenium, and vitamins B1, B6, E, and K. Among these crops, pea and French bean are grown widely as pulse vegetables. Peas are affected by a number of bacterial, fungal, viral, phytoplasmas and nematode diseases. These diseases, under the right conditions, can significantly decrease both yield and quality. Important groups of organisms causing soilborne diseases on pea include fungi, bacteria, and nematodes. Soilborne fungal diseases include seedling diseases, root rots and wilts and soilborne nematode diseases like pea cyst, the root knot and the root-lesion nematodes. Foliar diseases caused by fungal pathogens include white mold, powdery mildew, downy mildew and Ascochyta blight and bacterial diseases include bacterial blight as well as brown spot and major diseases caused by viruses include *Alfalfa mosaic virus*, *Bean leafroll virus*, *Pea enation mosaic virus*, *Pea streak virus*, *Red clover vein mosaic virus* and *Pea seedborne mosaic virus*. In case of beans, bacterial wilt, common blight, Fusarium root rot., Fusarium wilt., halo blight, rust and white mold, golden mosaic diseases affect the production and quality of the produce significantly.

Name of disease: ROOT AND STEM ROT

Geographical origin, distribution and crop losses: Globally, root rot is estimated to cause yield reductions of 10–30% in pulse crops, but losses can be as high as 100% in crops with severe infections under ideal environmental conditions.

Symptoms: Infection leads to drooping and wilting of plants at early stage of plant growth. In pea, symptoms appear as vascular discoloration of stem and reddish appearance in the pith extending towards roots. This is a characteristic symptom of *F. solani* in pea. Sometime general yellowing of entire plants



Fig. 44. Root and stem rot symptoms on beans

is observed in cowpea due to Fusarium infection (Fig 44). *Rhizoctonia* infection in pea results drooping followed by wilting of the entire plants. The bark of collar region and root portion develops complete rots and degenerate. This is easily peeled off with slight pressure on bark leaving only pith portion. This is most destructive disease of cowpea. Symptoms appear on stem near the soil level and extend downward into the roots and upward also. Lesions are somewhat sunken and reddish brown. Later, as the lesions enlarge, they turn gray to ash colour at the center and numerous minutes, black dot like fruiting body developed from the center. The inner portion of stem pith becomes hollow. Dull white mycelial growth is observed near soil line at initial stage of infection in humid weather.

Causal organism: This disease is caused by *Fusarium solani*, *Fusarium oxysporum* f. sp. *pisi* and *Rhizoctonia solani*.

Key characteristics: The *Fusarium* spp. are mostly known in their asexual states. Both *F. solani* and *F. oxysporum* produce one- to five-septate, fusoid macroconidia, cylindrical to oval microconidia, and globose chlamydospores that are intercalary or borne on short lateral branches. *Fusarium solani* produces microconidia on long conidiophores, terminating in a single cylindrical to barely subulate phialide measuring 45 to 80 µm long, while those of *F. oxysporum* are produced on short conidiophores with numerous short, simple phialides.

Disease cycle and favourable conditions: *Fusarium* species survive in the soil as chlamydospores, which can remain viable for as long as 10 years. *Fusarium oxysporum* hyphae penetrate directly through the cortex into the vascular system, with little or no development of cortical lesions. *Fusarium solani* hyphae generally penetrate through the stomata of the epicotyl and hypocotyl, although direct penetration through the cuticular surface of pea epicotyls also occurs.

Management

- Long crop rotation with wheat and bajra.
- Field sanitation by uprooting and burning of all infected plants.
- Summer fallowing, ploughing accompanied with one irrigation followed by again ploughing in summer to reduce the soil inoculum.
- Green manuring in June and July followed by soil application of *Trichoderma* @5 kg/ha soon after ploughing of the sun hemp.
- Seed treatment with *Trichoderma* @0.6 to 1% depending upon soil and pathogen status.
- Drenching of *Trichoderma* @1% suspension after 20 days of sowing in beans only.
- Maintain proper drainage and aeration in the field.
- Apply balanced dose of fertilizers including micronutrients in soil.

References:

Pandey S, Mishra RK, Mishra M, Parihar AK, Dixit GP (2021) Recent Approaches for Diagnosis and Management of Economically Important Diseases of Field Pea (*Pisum sativum* L.) in India. *Innovative Approaches in Diagnosis and Management of Crop Diseases: Volume 2: Field and Horticultural Crops*.

Name of disease: COLLAR ROT

Geographical origin, distribution and crop losses: The fungus attacks many hosts and is present worldwide. No loss assessment reported in case of pea and beans.

Symptoms: Initially wet rotting of bark is observed. Rotting covers entire bark of the plant near collar region. Characteristic symptoms are observed as white, fungus growth on affected portion as well as on contact soil (Fig. 45). Gradually this hyphal mat is converted into small, mustard like sclerotia that survives in the soil.

Causal Organism: The disease is caused by *Sclerotium rolfsii* and is becoming ubiquitous with broad host range.

Key characteristics: The fungus produces white fungal strands (mycelia or hyphae) around infected plant parts and can be observed on the soil surrounding the plant. They generally form small, tan to dark-brown/black, spherical sclerotia, that function as survival structures.

Disease cycle and favourable conditions: Sclerotia are the principal overwintering structures and the primary inoculum source for the disease. Under favorable conditions, sclerotia germinate and fungal hyphae grow towards and attack the lower part of the stem base. On diseased tissues, a hyphal mat and sclerotia are produced and, sometimes, also basidiospores are produced. The role of basidiospores in the disease cycle under field conditions has not been investigated in detail.

Management:

- Crop rotation with low land paddy and other cereals.
- Field sanitation by uprooting and burning of all infected plants.
- Summer fallowing, ploughing accompanied with once irrigation followed by again ploughing in summer to reduce the soil inoculum.
- Green manuring in June and July followed by soil application of *Trichoderma* @5 kg/ha soon after ploughing of the sun hemp.
- Seed treatment with *Trichoderma* @ 0.6 to 1% depending upon soil and pathogen status.
- Remove all the weeds from field.
- Drench *Trichoderma* @1% suspension after 20 days of sowing to reduce early infection in beans.

Name of disease: POWDERY MILDEW

Geographical origin, distribution and crop losses: Its occurrence has been reported from Afghanistan, Alaska, Argentina, Australia, Canada, China, France, Germany, Hungry, Italy, Iraq, Japan, Libya, Malaysia, Mauritius, Pakistan, Philippines, Portugal, Russia, Spain, Siberia, South Africa, Sudan, Switzerland, Tanzania, Tucuman, Turkey, U.S.A., Zambia and many other countries. In India, the disease was first reported by Butler (1918) from Dehradun (U.P.). But reports of its common occurrence all over the pea growing areas of the country have been received only after 1935. Munjal et al. (1963) reported that in a completely infected crop, the losses were 21–31 per cent in terms of pod number and 26–47 per cent in terms of pod weight. However, about 50 per cent reduction in yield due to attack of this pathogen.

Symptoms: Symptoms appear in month of February as white to light gray, powdery fungal spores on leaves, twigs, tendrils and pods as pustules (Fig. 46). Soon these pustules coalesce and cover all foliar part of pea. Infection of pods leads partial filling of the grain and pod remain smaller in size. Outer skin of infected pods becomes gray in colour and rough.



Fig. 45. Growth of *Sclerotium rolfsii* on stem and soil contact

Causal organism: This is a serious disease of pea and caused by *Erysiphe pisi*.

Key characteristics: The fungus is ectoparasitic, spreading on the surface of the host and sending haustoria into the epidermal cells to draw out nourishments. The haustoria lie outside the cytoplasm of the host and special structures are formed between the haustoria and the cytoplasm. These interfacial structures distinguish haustoria from hyphae and therefore, their structures and physiology are important factors in parasitism. The conidiophores arise vertically from the superficial hyphae on the host surface. Each conidiophore bears conidia either singly or in chains. Of these, only the last one in the chain matures as the spores are formed in regular order from the tip downwards. The mature conidia fall-off quickly and are disseminated by wind.



Fig. 46. Symptoms of powdery mildew on pea

Disease cycle and favorable conditions: The fungus is an obligate parasite and perennates either through cleistothecia. The primary infection of the lower leaves is caused by windblown ascospores (where ever cleistothecia are present) or by conidia formed on the crop sown earlier in the neighbouring areas. The conidia formed abundantly on primary infections are blown by wind and air currents and cause secondary infections. The powdery mildew of pea becomes a serious problem under warm and dry conditions during the day and cool nights. Irrigation at flowering to pod formation stages can prolong the vegetative phase, thereby increasing period for susceptibility. The disease is more prevalent in dry weather and moderate temperatures. A temperature range of 10-30°C is favorable for conidia germination with optimum being at 20°C.

Management

- Avoid delay in sowing and must complete up to first fortnight of October.
- Foliar sprays of carbendazim 50% WP @ 250 g per hectare alternated with sulphur 40% WP@5.65 Kg per hectare or sulphur 52% SC @ 2 lit per hectare, or Sulphur 85% DP@ 15-20 Kg per hectare, Benomyl 50% WP @ 200 g per hectare, Dinocap 48% EC @ 300g per hectare, Fenarimol 12 % EC @ 0.05%, Infected crop debris must be burnt after harvesting of pods.

References:

Ullasa B, SI S (1980) Field Resistance of Pea Germ Plasm to Powdery Mildew (*Erysiphe polygoni*) and Rust (*Uromyces fabae*). Plant dis 1085.

Name of disease: Rust

Geographical origin, distribution and crop losses: The disease is a serious menace to the cultivation of this crop in wet areas the world over. The identity of the fungus was established long back by Jordi in 1904 (Buchheim, 1922). Since then it has been reported from Morocco, U.S.S.R, Burma, France, Germany, Japan, U.S.A., Afghanistan, Palestine, Switzerland, Norway, Portugal, Pakistan, Lithuania and Israel. In India, pea rust pathogen (*I. viciae fabae*) was first reported on *Vicia faba* by Sydow and Butler from Pusa (Bihar) in 1906. The first report of its occurrence on pea in India was by Butler (1918). Subsequently the disease was reported from Purvanchal where it caused severe damage to sweet pea crop and now is prevalent in severe form in the North and North Western part of India. Pea rust is an important disease, which occurs quite frequently in pea growing areas in North and North West parts of India.

Symptoms: Characteristic symptoms observed as minute, slightly raised pustules on all above ground plant parts (Fig. 47). Generally elongated pustules are seen on stem. Rust pustules are never observed on pods of pea. These pustules on pea are distinct, faint yellow, circular sori consist of numerous uredospores. Later, dark red colored teleutosorous are formed on the leaves and tendrils. Uredopustules are larger in size and reddish brown as compared to teleutopustules. Early bloom stage of crop to four weeks before harvest is most critical period for rust and must be protected from the infection.

Causal organism: The causal agent of pea rust is *Uromyces fabae*, *U. pisi*, while rust of cowpea is caused by *U. phaseoli*.

Key characteristics: *U. viciae-fabae* is autoecious rust with all its spore stages on the same host and no alternate host is required in the life history. The fungus is heterothallic. On peas the pycnia occur in small groups associated with the aecia. The peridium is short and whitish. The aeciospores are round to angular or elliptical with hyaline wall. The wall of these spores is verrucose. The uredia present a powdery appearance. The uredia present a powdery appearance. The telia occur in the same sorus as the uredia. They are dark brown to black. The teliospores are subglobose, ovate or elliptic with rounded or flattened apex.

Disease cycle and favorable conditions: In India the rust appears to survive on weed hosts belonging to *Lathyrus*, *Vicia* etc. and the spores are windblown to the main crop. Wild hosts may serve as primary or secondary source of infection. Aeciospores in *U. viciae-fabae* were found to be repeating spores and play an important role in pea rust outbreaks. The observed relationship between severity of pea rust and duration of leaf wetness at 20°C may be useful in predicting disease outbreaks if initial inoculum is present. Production of aeciospores was observed at a temperature range of 10-25°C, with a maximum at 25+2°C. A relative humidity (RH) of 100% was favorable for aeciospore germination while 98% RH favored urediospore germination.

Management

- Avoid delay in sowing of pea and must complete up to first fortnight of October.
- Fungicidal sprays should be done in critical period if an average of two pustules per leaf observed in the field.
- Foliar spray of lime sulphur 22% SC @1%, sulphur 80% WP @ 3.13 Kg per hectare, sulphur 85% DP @ 15-20 Kg per hectare, tridimefon 25% WP @ 0.1% in conventional sprayers.
- Burning of infected crop debris is very effective for reduction of inoculum.

Name of disease: LEAF BLIGHT

Geographical origin, distribution and crop losses: The disease was reported first time in Delhi, India and can be occurred in all parts of the humid tropical areas of Asia and many other countries and is prevalent in all parts of humid tropical areas of India, Bangladesh, Indonesia, Malaysia, Philippines, and Thailand. The disease causes qualitative and quantitative losses up to 96 per cent under natural epiphytotic conditions.

Symptoms: Symptoms appear as black, sooty and moldy growth of fungus on all foliar part (Fig. 48). Initially the spore mass are olivaceous dark brown and spread very fast in the field. Quick defoliation is characteristic feature of the disease, which leads to naked stem and twigs on the plant. Symptoms of *Cercospora* infection

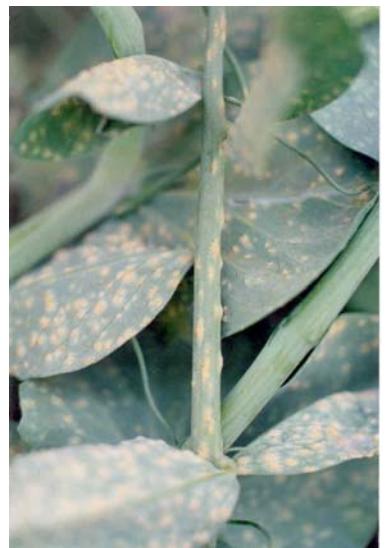


Fig. 47. Distinct, faint yellow, circular rust sori on pea

are observed as small circular brown spots with reddish margin.

Aschochyta blight is severe disease of cowpea, French bean and pea. *Aschochyta paseolorum* infects cowpea producing large, circular to irregular brown spots with reddish margin on leaves and all other plant parts.

Causal organism: *Pseudocercospora cruenta* and *Cercospora cruenta* both are separate species infecting cowpea. *Pseudocercospora* is more destructive than *Cercospora*.



Fig. 48. Leaf blight symptoms on cowpea

Key characteristics: Conidia of most *Cercospora* spp. are borne on erect conidiophores formed as fascicles above the leaf surface, which protrude into the air. The conidia are detached passively and are dispersed by wind or rain-splash. The principal agents for dispersal of conidia from infested residue on the soil surface are likely to be wind and rain, which influence spatial patterns of disease distribution.

Disease cycle and favourable conditions: The pathogen produces conidia in lesions of plant debris. These conidia play a role of primary inoculum in disease incidence. Rain splashes also play as a major role in dispersal of conidia. *Cercospora* leaf spot is considered as an important pathogen not only due to its widespread range but also due to the susceptibility of many commercial crops to this disease. Epidemiological conditions to produce conidia require 90-100 percent relative humidity and 20-26°C temperature. For germination and to cause the infection the ideal temperature recorded is 25-30°C. If the temperature is below than 10°C then no conidia are formed.

Management

- Avoid delay in sowing of *kharif* cowpea and completed by last week of June.
- Collect the defoliated leaves and burn in field itself to reduce the inoculum at source.
- There is no label claim fungicide recommended for this disease for pea and beans.

Name of disease: SCLEROTINIA BLIGHT

Geographical origin, distribution and crop losses: In India, the disease was first recorded in 1968 from Katrain area of Kullu valley of Himachal Pradesh. Besides Himachal Pradesh, the disease has also been reported from Uttranchal, Punjab and Kashmir. In India, 20.8 to 70.3 per cent loss in yield has been reported due to this disease. Due to extensive cultivation of peas during rabi season in northern plains and during summer in dry and wet temperate zones of Himachal Pradesh and Uttranchal.

Symptoms: This pathogen is having broad host range but proves very destructive on pea and French bean causing complete killing of the plants (Fig 49). Yield loss was recorded in these two crops up to 50% in North Bihar and Eastern U.P.



Fig. 49. Symptoms of Sclerotinia blight in pea and French bean

Symptoms are observed as wet, soft and white rotting of the tissues. Very soon white fungus growth is observed on the rotted portion. Later, embedded sclerotia in white mycelium formed on the infected portion as well as inner portion of pith and fruits. Primary infection on pea and cowpea always starts from floral infection followed by pods. Seeds converted into sclerotia and become concomitant mixture during threshing.

Causal organism: The disease is caused by a ubiquitous fungus *Sclerotinia sclerotiorum*.

Key characteristics: The mycelium of the fungus is hyaline, septate and branched. Hyphae are inter and intracellular and invade all the tissues of the affected host portion. The mycelium forms sclerotia, which are generally irregular and elongate and vary in size with age and environmental conditions. Apothecia emerge through pores in rind of the sclerotia when moisture is abundant. Apothecia are tan, fleshy, funnel-shaped cups on thin stalks. Ascospores ($5.9\text{-}7.3 \times 11.7\text{-}15.1 \mu\text{m}$) are discharged from the apothecia in great abundance to a height of 1-15 cm.

Disease cycle and favorable conditions: The pathogen perpetuates in the form of sclerotia left in soil or plant debris. The sclerotia germinate either carpogenically or myceliogenically. Sclerotia on or just below the soil surface can germinate myceliogenically and form mycelium, which invade plant issues directly at the contact points. During the extended periods of cloud and high moisture, ascospores discharged from apothecia are the more common source of infection. Ascospores either directly infect the host or they fall on fallen flowers (petals) and grow in them to produce mycelium. This mycelium can also cause infection and help in secondary spread of the disease. After infection, the fungus produces oxalic acid, which kills the cells in advance.

The fungus responsible for this disease is soil borne in the form of sclerotia, which can infect the susceptible host over a wide range of temperatures i.e. from 0 to 28°C with an optimum at 20 to 25°C . The fungus can tolerate wide pH range but is best adapted to an acidic substrate. Ascospores start germinating within 4 h at a temperature range of 5- 25°C (optimum 20°C). At $15\text{-}20^\circ\text{C}$, number and dry weight of sclerotia is maximum. Prolonged spells of rain during February-March play an important role in the primary infection and spread of the disease and the incidence was positively correlated with the percentage of rainy days and total rainfall.

Management

- All infected part must be carefully collected and burnt before drying of the plants.
- Seed cleaning is essential to remove the sclerotial mixture during threshing and processing.
- Deep ploughing, low land paddy with continuous stagnation of water reduces the sclerotial population.
- Close planting and dense canopy of plant should be avoided.
- Remove all the weeds particularly dicots from the field.
- Fungicidal spray must be started at early bloom stage or flowering stage. Alternate spray of carbendazim @0.1% and mancozeb @0.25% at 10 days interval in cool cloudy and wet weather.

Name of disease: BACTERIAL BLIGHT

Geographical origin, distribution and crop losses: In India, the first report of the disease was made in 1972 at Udaipur, Rajasthan. Severe outbreak of this disease was observed during 1986-1987 crop season in Kullu valley of Himachal Pradesh. Subsequently, Jindal and Bhardwaj (1989) also noticed the disease in Solan, Kullu, Mandi and Sirmaur districts of Himachal Pradesh. It is also known to cause as much as 70 per cent yield loss in pea.

Symptoms: Common blight is the major disease of bean particularly in cowpea. Disease appear in the month of July to September in rainy season. Symptoms of common blight are first seen as small translucent, water- soaked spots on leaves. Symptoms of common blight, fuscous blight and brown blight are difficult to differentiate based on symptoms except, isolation of casual organism. However, the infected tissue may turn yellow and die to form lesions of various sizes and shapes. Bigger blotch symptoms appear on the leaves in severe case of common blight particularly in rainy season (Fig. 50). Small spots appear on pods of different ages. Vascular discoloration is evident in severe incidence. Halo blight has most of the symptoms of common blight along with a chlorotic halo.

Causal organism: The pathogens associated with bacterial blight are *Xanthomonas phaseoli* pv. *phaseoli* (common blight); *X. citri* pv. *fusca*s (Fuscous blight), *Pseudomonas syringae* pv. *phaseolicola* (Halo blight), *P. syringae* pv. *syringae* (Brown spot blight).

Key characteristics: Races of the bacterium on pea were first distinguished by the differential reactions of the pea cultivars, Early Onward and Patridge. One hundred and forty-six isolates of *P. syringae* pv. *pisi* from UK and overseas were categorized into six races using a set of differential. *P. syringae* pv. *pisi* consists of seven identified races. One of these (Race 6) lacks all avirulence genes and is common around the world and in Australia. Globally, Race 2 and Race 6 predominate; however, in Australia, Race 3 predominates due to the widespread cultivation of cultivars susceptible to Race 3, but resistant to Race 2.

Disease cycle and favorable conditions: Neither soil nor infected plant parts are primary sources of infection in any of the pea growing areas. The pathogen overwinters in the infected seed. The seed carries the bacterium both externally and internally. The bacterium can persist in the infected seed for three years. It colonizes the intercellular and intracellular spaces of the seed coat but does not penetrate the embryo or cotyledons. Even a very low level of seed infection can cause economic loss, since the disease can spread fast from primary infection foci. The pathogen is carried in irrigation water, splashed by rain or blown in wet winds to other plants and field infection usually occurs through stomata and wounds. The optimum temperature for this disease is 27.7°C, while minimum is 7.2°C and maximum 37.7°C. Frost is an important factor affecting disease development.

Management

- Use of clean, disease free and certified seeds.
- Crop rotation of fort two years to dispose-off over wintering bacteria.
- Field sanitation by burning of all the infected leaves and crop debris.
- Proper drainage should be maintained in the field to avoid water stagnation.
- Sowing time should be adjusted in such a way that fruiting stage should not coincide with heavy rainy period.



Fig. 50. Symptoms of bacterial blight on cowpea

PHYTOPLASMA ASSOCIATED DISEASES WITH COWPEA

***Vigna unguiculata* L. (Cowpea):** Phytoplasma strains infect cowpea crops and cause serious losses all over India.

Symptoms: Witches' broom, Shoot proliferation and flat stem (Fig. 51).

Distribution: All over India

Causal organism: UP: 16Sr I-B & VI-D; TN: 16Sr I & II; Kerala: 16Sr II-D; Andhra & Telangana: 16Sr II; Haryana: 16Sr II-D; Karnataka: 16Sr VI-D; Maharashtra: 16Sr I & II; Tripura: 16Sr I & VI; Delhi: 16SrII-D.



Fig. 51: Witches' broom, shoot proliferation and flat stem symptoms of cowpea

Cucurbits

Introduction

Cucurbits have very important place in vegetable production. It remains available for about 8-10 months in the year. Cucurbits belong to the family Cucurbitaceae having a large family of plants with 130 genera and 800 species known all around the world and among them and out of that it contains at least 9 genera and of them 16 species are cultivated such as sponge gourd, ridge gourd, muskmelon, watermelon, round melon, pumpkin, vegetable marrow cucumber, snake gourd, pointed gourd, kheera, bottle gourd etc. They have nutritional and medicinal value. Cucurbitaceae members have been characterized with insecticidal/ wormicidal control, anti-hypoglycemic, anti-inflammatory, anti-lipidemic characteristics. Fruits of bottle gourd and bitter gourd are used for control of diabetes. Cucurbits are warm weather crops which are sown, grown and harvested over spring, summer and autumn. The cucurbit crops suffer from several fungal, bacterial, viral, nematodes and nutrients deficiency diseases. Among them, fungal diseases [powdery mildew (*Podosphaera xanthii* and *Erysiphe cichoracearum*), downy mildew (*Pseudoperonospora cubensis*), anthracnose fruit rot (*Colletotrichum orbiculare*), damping off and fusarial wilt (*Fusarium oxysporum f.sp. niveum*)], bacterial diseases [bacterial wilt (*Erwinia tracheiphila*), bacterial fruit rot (*Pectobacterium* sp.) and angular leaf spot (*Pseudomonas syringae* pv. *lachrymans*)], viral diseases (cucumber mosaic virus and pumpkin mosaic virus) and nematodes diseases (root knot of cucurbits and Reniform nematode) are major diseases to reduce the production and quality of the produce of cucurbits.

Name of disease: DAMPING OFF, SEEDLING AND ROOT ROTS OF CUCURBITS

Geographical origin, distribution, and crop losses: Damping off and root rot of cucurbits has been reported from nursery beds and young seedlings of cucumber, melon, watermelon, pumpkin, summer squash, and winter squash throughout the cucurbit growing areas. In green houses, cucumber damping off disease is widespread. These diseases have increased rapidly under protected cultivation.

Symptoms: Water-soaked lesions occurring at soil level are the first visible symptoms of damping off in cucurbitaceous family. The disease spreads and ultimately the seedling wilts and decays. Seedlings may die in the soil itself before emergence or may be attacked when the cotyledonary leaves are opening. After emergence of the seedlings



Fig. 52: Pythium Damping off of cucumber at seedling stage and cottony leak of cucumber fruits caused by *Pythium* spp.

from the soil surface, water-soaked lesions appear at the collar region and seedlings droop after 2 to 3 days which is symptomatic of post-emergence damping-off (**Fig. 52**). Often, plants that have survived damping-off may show symptoms of root rot. Root rot affected roots shows a watery grey appearance. Cottony leak, sometimes called *Pythium* fruit rot or *Pythium* cottony leak, is a common disease of cucurbits which appears in the form of white cottony growth of the pathogen on the fruit (**Fig. 52**).

Causal organism and Key characteristics: Cucurbit damping-off and seedling root rots have been reported to be caused by various species of *Pythium* like *P. aphanidermatum* (Edson) Fitzp., *P. irregularare* Buisman, *P. ultimum* Trow. and *P. spinosum* Swada. *Rhizoctonia solani* Kuhn, *P. capsici* Leon. and *Phytophthora drechsleri* Tucker occur independently and in combination with the species of the *Pythium*, *Fusarium* spp. including *F. oxysporum* f. sp. *cucumerinum* Owen frequently causes rotting of seeds and damping-off.

Disease cycle and favourable conditions: Damping-off of cucurbits is usually most severe in conditions having high soil moisture, high plant population, lack of aeration, cool, damp and cloudy weather. In addition, root rot is also favoured by deep planting. Severe incidence of *P. aphanidermatum* was found in watermelons and muskmelon which were sown in cool, moist soil which appears to form crusts around or over the hypocotyls. Soon after one week of sowing or transplanting seedlings get more prone to damping off. Unsterilized soil in green houses is more susceptible to damping off fungi and heavy irrigation also exacerbates damping-off. *Pythium* fruit rot is favored due to moist soil at growing time.

Disease management

Cultural practices:

Soil solarization and soil enrichment with organic matter or oil cakes and or biofumigation have a great potential to prevent incidence of damping off. Biofumigation with cabbage residue incorporated at the top 20 cm of soil at the rate of 5 kg m⁻² and solarization enhanced crop growth. Effects on pathogen inoculum levels, disease severity and plant growth parameters were greater during summer seasons than in winter. Simple cultural practices like sanitation measures, irrigation schedule, raised beds and good quality seeds help to keep the disease away.

Chemical control:

Seed treatment with captan or thiram @ 2.5 to 3 g/kg seed is found effective to control incidence of damping off disease. Drenching with the mixture of mancozeb (0.25%) and carbendazim (0.1%) to the plants after seedling emergence controls the disease significantly.

Biological control:

Application of biocontrol agents through seed treatment and soil application in the nursery bed before sowing is a good option to control the disease. Actinomycetes (*Streptomyces*) isolates inhibited the growth of the *P. aphanidermatum* in culture as well as under greenhouse conditions against damping off disease in melon. Cucumber seeds treated with spores of *Serratia marcescens* N4-5 and ethanol extracts of the bacterial cultures provide significant suppression of damping-off disease caused by *P. ultimum*. Besides cucumber, the spores of this bacterium also suppress damping-off caused by *P. ultimum* in cantaloupe, musk melon, and pumpkin. Selective antagonistic bacterium *Bacillus pumilus* strain SQR-N43 can manage damping off disease of cucumber caused by *R. solani*. The application of bioagent stabilizes the population and increases the active form of the antagonist. The treated cucumber seeds with ethanol extract of *Serratia marcescens* N4-5 controls damping off caused by *P. ultimum*. They also reported that N4-5 extract was found compatible with two *Trichoderma*

isolates and can be used as a seed treatment in combination with in-furrow application of *Trichoderma* isolates. A significant reduction (up to 70%) by *T. harzianum* has been reported to be effective against damping-off in cucumber caused by *P. aphanidermatum* under field conditions.

Soil biofumigation by incorporation of mustard crop in soil at flowering stage and soil cover with plastic for ten days is commonly used by farmers for managing many soil borne problems including cucumber damping-off. Soil amendment with canola residue is very efficient in consortial bioformulation, a mixture of *Trichoderma harzianum* strain T969 and the fungicides Metalaxyl and Metalaxyl MZ when applied into soil medium at a CFU of 10^7 conidia ml/L is effective in controlling damping-off disease caused by *P. aphanidermatum*, *P. ultimum*, *P. irregular* in polyhouses. Use of medium with spent blewit peat compost (SBPC), consisting of 50% (v/v) SBMC, 0.3% (w/v) lime and 50% (v/v) BVB has been Pythium damping off in cucumber seedlings. The SBPC enhances plant growth promotion of cucumber seedlings and reduces disease incidence. Among these, *Bacillus aryabhattai* CB13 effectively suppressed *P. aphanidermatum* in the steamed SBPC medium. Steamed SBPC treated cucumber plants show 58% disease incidence but there was suppression up to 4% in the bio-formulated container medium. This new bioformulation showed high potential to control Pythium damping-off in commercial nurseries.

Name of disease: ANTHRACNOSE

Geographical origin, distribution and crop losses: The disease was first time reported in 1867 on gourds from Italy. It occurs mainly in all humid regions of the world, where the crop is grown. In India, the occurrence of the disease has been reported from Punjab, Haryana, Assam, Karnataka and other states wherever cucurbits are grown in rainy weather Ullasa and Amin (1986) reported yield losses of marketable fruits up to 99.5 per cent due to this disease.

Symptoms: Anthracnose is a common problem of most of the cucurbits. Disease symptoms are found on all above ground plant parts started from cotyledonary leaf to fruits. Symptoms on leaves are observed as water soaked, small yellow spots that enlarge and turn to brown. The necrotic portion dries and shatters (Fig. 53). Elongated water soaked, sunken lesions appear on stem. Light yellow to brown discoloration of these stem lesions is due to abundant sporulation. Severe incidence resulting infection of juvenile fruits appears as small, sunken, light brown, cracked spots. Two species are involved in cucurbits which is evident from the size of spots is distinct and appear in different weather condition.



Fig. 53. Necrotic spots on leaves and fruit of cucurbits

Causal organism: This disease is caused by *Colletotrichum orbiculare* (Berk. and Mont.) Arx (Syn. *C. lagenarium* (Pass.) Ellis and Halst. The teleomorph *Glomerella lagenarium* F. Stevens is rarely found in nature.

Key characteristics: The mycelium is septate, hyaline when young and dark when old. Stromata (acervuli) are brown to black and variable in size bearing black setae and hyaline conidiophores on the host surface. Setae are brown, thick-walled, 2-3 septate, and 90-120 μm long. Conidia are produced one at a time at the tip of the conidiophores and accumulate in a slimy, pinkish mass. Individually the conidia are hyaline, oblong to ovate oblong, 1-celled, and measure 13-19 $\mu\text{m} \times 4-5 \mu\text{m}$.

Disease cycle and favorable conditions: The pathogen is mainly soil borne but it is become seed borne, when the fruits are infected, and mycelium reached to the seed. The pathogen can also survive in infected plant debris in the field and in volunteer plants and cucurbit weeds. Infected crop residue and infected seeds, volunteer plants and cucurbit weeds are primary sources of fresh infection. The disease spreads by windblown rain, people, animals, and machinery moving through the crop in wet or moist conditions. In favorable weather conditions conidia are disseminated to the healthy plants and cause infection. The spores of the pathogen are germinated best at 22-27°C and relative humidity is 100 per cent for at least 24 h. Humid rainy water is essential for infection. If the moisture is present, spores germinate, and penetration occurs effectively up to 72 h after conidial deposition and fruiting bodies of the fungus are visible on the lesions in next few days. The disease development can occur at 20-30°C with optimum temperature at 25°C and 100 per cent relative humidity for at least 18 hours.

Management

- Always use seed collected from healthy fruits and disease-free area.
- Use disease-free and certified seeds
- Seeds treatment by carbendazim @0.25%.
- Field sanitation by burning of crop debris.
- Grow crop on bower system to avoid soil contact.
- Maintain proper drainage in the field.
- Seed production should preferably carry out in summer season because summer crop is often free from pathogen.
- Foliar sprays of carbendazim 50 0 % WP @300 g per hectare, zineb 75% WP @ 1.5 to 2 Kg per hectare started soon after infection.

References:

Nagendran K, Kumari S, Dubey V, Pandey KK (2020) Development of integrated disease management (IDM) module for major diseases in bitter gourd. Vegetable Science, 47(1), 39-43.

Name of disease: DOWNTY MILDEW

Geographical origin, distribution and crop losses: Downy mildew disease was first reported from Cuba in 1868 (Chupp and Sherf, 1960). It is prevalent in the warm temperate and tropical regions like North America, Europe and Asia. In India, it is present all over the country except in temperate zone in high altitude of the Himalayas. The disease is very common in North India particularly during later parts of rainy seasons. Mahrishi and Siradhana (1988) reported that musk melon fruit yield reduction was 79.06 per cent in Rajasthan under untreated crops.



Fig. 54. Symptoms of downy mildew on cucumber

Symptoms: Cucumber, bitter gourd, bottle gourd, sponge, gourd ridge gourd and muskmelon are severely affected by downy mildew. Symptoms appear as irregular, numerous, small, yellow areas surrounded by green tissues scattered all over the leaf lamina. It appears just like in definite mosaic pattern particularly in cucumber (Fig. 54). The yellow areas are angular and bounded by veins. Symptoms on bitter gourd is light brown while grayish brown on bitter gourd without prominent yellowing on these host. In high humid weather, faint white downy growth of fungus is observed.

Causal organism: This disease is caused by *Pseudoperonospora cubensis*.

Key characteristics: The mycelium is coenocytic and intercellular and gives rise to small, ovate, intracellular haustoria, which sometimes develop finger like branches. The sporangiophore is branched either dichotomously or intermediately between dichotomous and monochotomous branching habit. The sporangia are grayish to olivaceous purple, ovoid to ellipsoidal, thin-walled, and with a papilla at the distal end. They measure 21-39 x 14-23 µm. The germination of sporangia occurs by production of biflagellate zoospores which are 10-13 µm in diameter when in resting state. Oospores are not common in the species. However, in India presence of oospores on certain cucurbits has been reported from Madhya Pradesh, Punjab and Rajasthan.

Disease cycle and favorable conditions: The exact disease cycle in different regions of the country is not known. However, in Punjab, when winter temperatures are too low for the growth of cucurbits, the fungus perpetuates in the form of active mycelium on self-sown or cultivated sponge gourd growing in sheltered places during severe winters and also in open during milder winters. The pathogen can survive as both mycelia and sporangia on a wide range of wild host. It is transmitted by insects and other invertebrates. In saturated atmosphere, maximum sporulation occurs at 18-28°C. The day temperature of 25-30°C, night temperature of 15-21°C and RH > 75% favours the disease development, 20°C is optimum temperature for germination and infection.

Management

- Crop should be grown with wide spacing in well-drained soil.
- Air movement and sunlight exposure helps in checking the disease initiation and development.
- Bower system of cropping reduces the disease incidence.
- Field sanitation by burning crop debris to reduce the inoculum.
- Seed production should be preferably carried out in summer season because summer crop is often free from disease.
- Alternate foliar spray of azoxystrobin 23% SC @0.1%, zineb 75% WP @0.2% and azoxystrobin 4.8% w/w + chlorothalonil 40% w/w @0.3%, metiram 55% + pyraclostrobin 5% WG, cymoxanil 8% + mancozeb 64% WP @ 1500 g per hectare

References:

Bhardwaj DR, Gautam KK, Saha S, Nagendran K, Pandey KK, Singh AK, Singh B (2018) Mining the source of resistance for downy mildew and gummy stem blight in bottle gourd (*Lagenaria siceraria*) accessions. Indian J Agric Sci 88(5): 746-50.

Name of disease: POWDERY MILDEW

Geographical origin, distribution and crop losses: The powdery mildew is reported to occur on cucurbits since 1800. It is a widely occurring disease in cucurbits. In India, the disease is prevalent in almost all the states particularly in the warm and dry areas, where moisture is present as dew. Powdery mildew affects cucumber, musk melon, bottle gourd, squash, pumpkin and water melon.

Symptoms: Bottle gourd, bitter gourd and pumpkin are most severely affected by the disease. This is more severe in winter



Fig. 55. Powdery mildew on bitter gourd

season and green house crops (Fig. 55). Symptoms appear on all foliar part as white to dull white, powdery growth. This white growth quickly covers most of the leaf surface and leads to heavy reduction in photosynthesis area. Plant may wither and die. Growth of plant and fruits seized. Transpiration rate is very fast from infected leaves.

Causal organism: Two pathogens are associated with this disease i.e. *Sphaerotheca fuliginea* and *Erysiphe cichoracearum*. In addition to these two fungi, *Leveillula taurica* has also been found to be associated with powdery mildew symptoms.

Key characteristics: Perithecia of all three pathogens have indeterminate mycelial appendages. Conidia of *E. cichoracearum* singe celled, hyaline, barrel shaped, in chains. Perithecia contain a single ascus. The ascospores are single celled, hyaline, spherical, 6-10 per ascus. Conidiophores are unbranched and erect. Cleistothecia are globose, mostly 70-100 µm in diameter and dark brown/black in colour. Occurrence of two races in *E. cichoracearum* and 4 (0, 1, 2, 3) in *S. fuliginea* has been reported by Khan and Sharma (1993). Race 1, 2 and 3 occur in most of the cucurbit growing areas of the country. Race 3 is wide spread and infects most of the cultivated cucurbits while race 2 is restricted to green melon (*Citrulus vulgaris* var. *fistulosus*) and race 1 to sponge gourd (*L. cylindrica* or *L. aegyptica*) and other cucurbits.

Disease cycle and favorable conditions: Cucurbit powdery mildew can overwinter as active mycelium and conidia in sheltered situations on a number of volunteer or self-grown cucurbits. These may initiate the disease in the local hosts and from there the primary inoculum in the form of conidia might be wind to the main crop in the plains. The conidia germinate and cause direct penetration of the epidermal cells in which they produce haustoria. Incubation period is about 3-7 days. The conidia formed abundantly on primary infections are blown by wind and air currents and cause secondary infections and the cycle is repeated. The fungi can sporulate and cause infection in a very dry as well as wet atmosphere, but infection increase as the atmospheric humidity increase, heavy dew deposits favouring the penetration by germ tubes. The minimum and maximum temperatures for conidial formation and host penetration are 10°C and 32°C, respectively and optimum temperature being about 26 to 28°C.

Management

- Alternate foliar sprays of carbendazim 50%WP @300g per hectare and azoxystrobin 4.8% w/w + chlorothalonil 40% w/w SC@0.3%, benomyl 50% WP @ 200 g per hectare.

Name of disease: FRUIT ROTS

Geographical origin, distribution and crop losses: Fruit rot is common disease of cucurbits in India. It occurs in almost every locality during the rainy season due to excess moisture. It is not only field disease but market and transit disease also.

Symptoms: Fruit rot is a common problem of kharif-sown cucurbits. Whole crop blighted within three to four days. Initially water soaked, and soft rotting of the fruits start from lower portion, which are close to soil contact in Pythium fruit rot. In early morning white, fluffy, mycelium growth is observed on the affected portion (Fig56a). Rhizoctonia fruit rot is most severe in muskmelon showing rotting and cracking of infected portion of fruit (Fig. 56b). Phomopsis fruit rot on ash gourd was first time observed during cropping season of 2000. *Phomopsis cucurbitae* on ash gourd is the first record in world. Symptoms are mostly observed on matured fruits as comparatively dry rotting with characteristic pycnidia over it. Generally lower portion of fruits touching soil surface are affected (Fig 56c).



Fig. 56 (a-c). White fluffy mycelium on bottle gourd, symptoms of Rhizoctonia in musk melon, Symptoms of Phomopsis fruit rotting in ash gourd

Causal organism: *Pythium* and *Phytophthora* are major pathogens for this rotting. *Phytophthora cinnamomi* is causing rotting of vines, leaves and fruits of pointed gourd.

Disease cycle and favorable conditions: The main pathogens are warm weather fungi. They persist in soil as oospores and if moisture, temperature, and other suitable conditions are present they cause infection of the fruits on or near the soil. Infection is always facilitated by bruises and wounds on the fruit. This is common when the fruit are rubbing against soil particles or when there are insect bites. Once the cottony growth has appeared zoospores produced by this growth cause secondary spread of the disease. The fungi are present in the soil living in a saprophytic manner on dead organic matter. *Pythium* spp. are required warm weather conditions for growth. A high soil moisture, high temperature and juvenile tissues of the host are important factors determining the development of the disease

Management

- Avoid soil contact of fruit by using bower system of cultivation and staking of plant.
- Provide proper drainage in the field.
- Green manuring followed by soil application of *Trichoderma* @5 kg/ha in soil is very effective in checking most of the fruit rotting.
- Collect affected fruit and burn them to reduce primary inoculum.

Name of disease: GUMMY STEM BLIGHT

Geographical origin, distribution and crop losses: Gummy stem rot (black rot) was first described in 1891 on cucumber in France, musk melon in Italy and on watermelon in the USA. It causes the fruit rot in numerous species of cucurbits. The disease also affects stem and foliage on cucumber, musk melon and watermelon.

Symptoms: This disease is now becoming severe in muskmelon, bottle gourd and sponge gourd. Most of the hybrids are susceptible to the disease. Introduction of hybrids introduced this disease in all cucurbits growing area. Initially water-soaked area is observed on the stem near soil line (Fig. 57). Later, translucent gum like exudates released from the affected portion is deposited over it. Black dot like pycnidia is also observed on the affected bark.

Causal Organism: The disease is caused by *Didymella bryoniae* (teleomorph) and its anamorph is *Phoma cucurbitacearum*.



Fig. 57. Symptoms of gummy stem blight

Key characteristics: The pycnidial stage is highly pleomorphic. The fungus produces two spore stages, a sexually produced spore perithecia giving rise to ascospores and an asexually produced spore pycnidia giving rise to conidia. Perithecia and pycnidia can be found embedded in the same lesion.

Disease cycle and favorable conditions: The disease is seed borne which is primary source of gummy stem blight inoculum. Infected crop trash, soil, weeds and infected seed are source of infection. Ascospores serve as the primary inoculum and are readily spread from field to field by wind and air currents. Conidia are released in a gummy substance and are therefore more adapted for short-distance movement through splashing water, which leads to secondary spread of the disease. Dark pseudothecia may also form, especially on stems, but are rarely seen. The optimum conditions for the infection process are temperatures ranging from 61°F-75°F and a that can survive on seeds, weeds (citron, balsam pear, and other volunteer cucurbits), and plant debris from previously infected cucurbits.

Management

- Avoid exotic hybrids and varieties due to high degree of susceptibility.
- Summer ploughing and green manuring followed by *Trichoderma* application.
- Maintain proper drainage and aeration in the field.
- Seed treatment with carbendazim @0.25%
- One spray and drenching of Carbendazim @ 0.05% near collar region.

References:

- Bhardwaj DR, Gautam KK, Saha S, Nagendran K, Pandey KK, Singh AK, Singh B (2018) Mining the source of resistance for downy mildew and gummy stem blight in bottle gourd (*Lagenaria siceraria*) accessions. *Indian Journal of Agricultural Sciences*, 88(5), 746-50.
- Pandey KK (2015) Recent advances in diagnosis and management of diseases of vegetable crops in India. Recent Advances in the Diagnosis and Management of Plant Diseases, 225-242.

Name of disease: LEAF SPOTS

Geographical origin, distribution and crop losses: In the tropics and subtropics regions throughout the world this disease is prevalent. It may become a serious disease if weather conditions are favourable. In Europe, this is a destructive disease of greenhouse crops.

Symptoms: The characteristic symptoms are the appearance of the water-soaked areas on the leaf lamina. These spots enlarge rapidly to become circular to irregular spots with pale brown, tan or white centers and wide purple to almost black margins. Didymella leaf blight was first time observed on ash gourd during 1998-99 and first report from India. Thereafter severity of the disease is increasing every year and engulfing ridge gourd, bottle gourd, pointed gourd, pumpkin and sponge gourd. Hybrids and exotic materials of bottle gourd and ridge gourd are highly susceptible to Didymella leaf blight. It is spreading through out the cucurbits growing area. Papery with rhythmic large spots followed by shot hole is observed in *Didymella* (Fig. 58a). Black small dot like fruiting structures are also observed on the old spots. White fungus growth is clearly visible on outer margins of the spots in morning periods. Several other types of leaf spots occurred on different cucurbits. Often these leaf spot diseases are more pronounced at maturity stage. Circular spots with variable size are observed on the leaf lamina. They are light brown to dark brown with white centre in *Cercospora* (Fig. 58b).

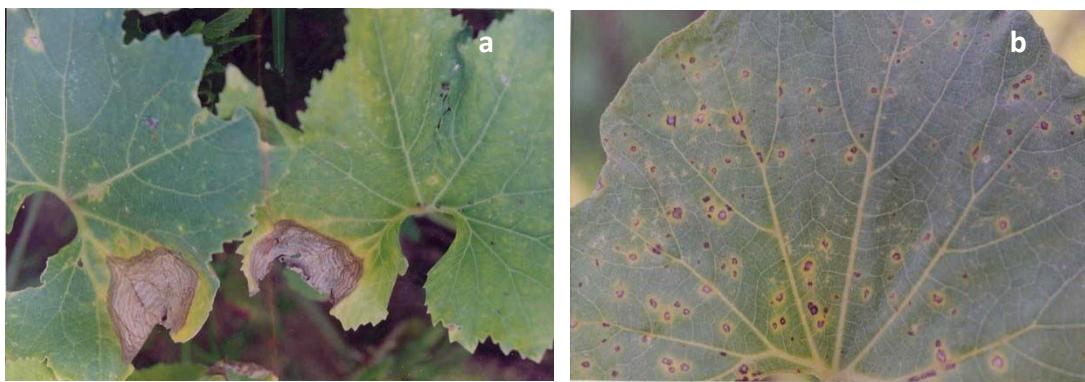


Fig. 58(a-b). Symptoms of Didymella leaf blight and cercospora leaf spots

Causal organism: The associated pathogens with this disease are *Cercospora citrullina*, *Didymella bryoniae*, *Alternaria cucumerina* and *Corynespora melonis*.

Key characteristics: The mycelium of *Cercospora* spp. is branched, septate and hyaline when young becoming coloured with age. The cells are multinucleate. Hyphae aggregate below the stomatal cavity to form stromata, which may be sclerotium like. These stromata enlarge and partly emerge on the host surface. The conidiophores are light brown to dark, septate, geniculate and bear conidia, which are hyaline, usually cylindrical and septate. Another fungus, *Corynespora melonis* (Cooke) Lindau causes similar leaf spots on cucumbers.

Disease cycle and favorable conditions: The fungi perpetuate on the perennial weeds and on the disease crop debris in the form of stromata. Conidia are formed. on these structures in the next season and are brown to long distances by moist wind. New lesions develop within 7-10 days of penetration by germ tubes from conidia. High humidity and 26-30 °C temperature are required for reproduction and infection. The disease is favored by high temperature and intense light.

Management

- Use healthy, disease-free and certified seed.
- Field sanitation and crop rotation reduces disease incidence.
- Fungicidal sprays of zineb 75%WP @0.2% alternated with carbendazim @0.05%
- Seed production should be preferably carried out in summer season because summer crop is often free from disease.

Name of disease: BACTERIAL WILT

Geographical origin, distribution and crop losses: This is an important bacterial disease problem and often poses threat to melon and cucumber production in several countries (Latin, 1996). Wilt has been reported in Europe, South Africa, Japan, United states of America and Canada. It is not a serious problem in squash, pumpkin and water melon.

Symptoms: The first signs of wilt appear usually on individual leaves as drooping, which become flaccid in sunny weather. As the disease progresses, more leaves wilt and eventually an entire plant is wilted (Fig 59). The wilting then becomes permanent, and the leaves and vine die. When wilted stems are cross-sectioned, viscous and sticky bacterial matrix exudates from the vascular bundles appears. This feature is used as a means of diagnosis. This is a vascular pathogen and differ from other *Erwinia* spp. is unable to degrade middle lamella. The bacteria over winters in the bodies of adult cucumber beetles particularly red striped and spotted

beetle. Primary infection is produced when beetles feed upon young leaves or cotyledons. The bacteria present in the vessels of infected plants die within 1 or 2 months after the dead plants dry up.

Causal Organism: Bacterial wilt caused by *Erwinia tracheiphila* is a common and often destructive disease on cucumber, muskmelon, squash and pumpkin.

Key characteristics: It is a gram-negative, rod-shaped, motile bacterium with peritrichous flagella. It is transmitted by insect vectors, primarily the striped cucumber beetle (*Acalymma vittatum* (Fabricius)) and the spotted cucumber beetle (*Diabrotica undecimpunctata howardi* Barber). The western spotted cucumber beetle (*D. undecimpunctata undecimpunctata* Mannerheim), the banded cucumber beetle (*D. balteata* LeConte), and other insects that cause wounds, such as grasshoppers, may also transmit the bacteria.

Disease cycle and favourable conditions: Not much is clear about the overwintering of the bacterium causing wilt. Earlier, it was thought to overwinter in the intestine tract of adult cucumber beetles on fence bushes or nearby trees. It has been observed that the bacterium remains viable in dried plant debris for very short time. Seed or soil does not transmit it. In the stems of host plant, the bacterium dies as the stem dries up and is disintegrated. *E. tracheiphila* remains viable in dried plant debris for very short periods of time. In plant stems, the pathogen dies as the stems deteriorate. It is not seed-transmitted, and there is no evidence that it survives in soil. Spread of bacteria to healthy shoots takes place through the dissolved walls of adjacent xylem vessels. Subsequent infections result from infected plants serving as inoculum sources.

Management

- Control of cucumber beetles at initial stage from the soil with neem cake or insecticides.
- Resistant varieties of cucumber should be grown.
- Summer ploughing of soil to expose all the stages of beetles.

Name of disease: MOSAIC AND LEAF DISTORTION

Geographical origin, distribution and crop losses: The virus has wide occurrence throughout the world and is known to infect about 800 plant species. Doolittle (1916) and Jagger (1916) described this disease for the first time. Since then it has been reported from various countries of the world, but it is more prevalent in temperate regions of the world. In India also, this disease has been reported. The infection of cucumber plants at cotyledonary stage reduced the yield by 89 per cent in summer and 96 per cent in winter.

Symptoms: Most of the cucurbits grown in rainy season are affected by virus diseases like cucumber mosaic, green mottle, leaf distortion, water mosaic etc. Generally alternate green and yellow patches with mottling symptoms are observed (Fig 60a). Sometime leaf deformed and curled downward. Smalling and narrowing of the leaves are also observed in leaf distortion virus (Fig 60b). Plants become small and excessive branching, proliferation of the leaves, twigs, petioles, bushy appearance of the plants are observed (Fig 60c).

Causal organism: The disease is caused by *Cucumber mosaic virus* (CMV) is a member of cucumovirus group and several strains, pathotypes and serotypes of this virus are known most of which can induce severe symptoms in cucurbits (Francki *et al.*, 1979). Based on serological studies, the virus is related to peanut stunt virus.



Fig. 59. Symptoms of bacterial wilt in muskmelon



Fig. 60 (a-c). Mosaic and green mottle, leaf distortion in cucurbits

Disease cycle and favorable conditions: The virus is transmitted in a non-persistent way by several aphid species including *Myzus persicae* (Sulzer), *Macrosiphum euphorbiae* (Thomas), and *Aulacorthum solani* (Kaltenbach). Acquisition of the virus by aphid instars takes place within one minute. The vectors lose their ability to transmit the virus within a period of two hours. Several factors such as vector type, virus strain, environmental conditions etc. affect the transmission efficiency. The virus is easily transmitted mechanically. There are divergent views on the seed-borne nature of CMV.

Management

- Management of the disease involves destruction of diseased hosts, weeds.
- Virus free and certified seeds should be used to check the seed transmission.
- Initial rouging of the infected plants.
- Periodical spray of systemic insecticides up to flowering stage to control vectors.
- Seed production should be preferably carried out in summer season because summer crop is often free from virus infection.

PHYTOPLASMA ASSOCIATED DISEASES

Cucurbita pepo L. (Pumpkin)

Symptoms: Witches' broom and stunting (Fig. 61)

Distribution: New Delhi

Causal organism: Aster yellows/16SrI-B (Rao et al., 2017)

Disease cycle and Epidemiology: This phytoplasma strain is widespread all over India



Fig. 61: Little leaf and witches' broom of *C. pepo*

***Lagenaria siceraria L.* (Bottle gourd)**

Symptoms: Little leaf, shoot proliferation and phyllody ([Fig. 62](#))

Distribution: Pune (Maharashtra) ([Tripathi et al. 2020](#)), Bengaluru ([Aswathappa et al. 2020](#))

Causal organism: *Ca. P. asteris*, aster yellows group (16Srl-B)

Disease cycle and Epidemiology: Not known



Fig. 62: Symptoms of little leaf (a) and (b) witches' broom on bottle gourd

Momordica charactia L.

Symptoms: Little leaf ([Fig. 63](#))

Distribution: Madikari (Karnataka); Pune (Maharashtra), Tripura.

Causal organism: Aster yellows group (16Srl-B) ([Venkataravanappa et al. 2017](#); [Verma et al. 2020](#)).

Disease cycle and Epidemiology: Reservoir of the identified groups of phytoplasmas in nature



Fig. 63. Symptoms of phytoplasma associated diseases in bitter gourd

Cole crops

Introduction

Brassicaceous or cruciferous vegetables, often called “cole” crops, are generally grown for their leaves and curd. These are cool-season crops, and tolerate light freezes and even brief heavy freezes, but prolonged deep freezes are fatal. Cole crops (*Brassica oleracea* varieties) is one of the most important groups of vegetable crops mainly cabbage, cauliflower, knolkhol, Brussels sprouts and sprouting broccoli. Among them, cabbage and cauliflower are popular and widely grown throughout the country. This group constitutes are widely grown on commercial scale while knolkhol, Brussels sprouts and sprouting broccoli are grown scattered on small scale. Cole crops known for their nutritional benefits because they are high in carotenoids, vitamins A and C, calcium, iron, magnesium, and dietary fiber. They also contribute a substantial amount of protein to the diet. Broccoli and kale are rich in carotene (provitamin A). Several volatile sulfur compounds are responsible for the characteristic flavor of the cole crops. These crops are infected by several diseases, leading to reduced to quantity and quality of the produce. Damping-off (*Pythium* species), downy mildew (*Hyloperonospora brassicae*), *Alternaria* blight (*Alternaria brassicicola* and *A. brassicae*), *Sclerotinia* rot (*Sclerotinia sclerotiorum*), club rot (*Plasmoidiophora brassicae*) and black rot (*Xanthomonas campestris* pv. *campestris*), individually or in combinations have inflicted heavy damage to seed industry and commercial vegetable production. To elicit such information and improve management strategies it is important to update information on the biology, epidemiology and management aspects of various pathogens causing diseases on cole crops.

Name of disease: DAMPING OFF OF CABBAGE AND CAULIFLOWER

Geographical origin, distribution and crop losses: The disease is of worldwide occurrence primarily in those regions, having moderate temperatures and high soil moisture. In India, it is prevalent throughout the country causing one or the other important diseases. In nursery, the pathogen, often causes more than 50 per cent seedling mortality. In some regions of Himachal Pradesh, the incidence was more than 50 per cent during March-April. The incidence was more in hybrid Royal Saluis than the varieties Snowball 16 and PSBK 1. In Kullu valley, up to 60 per cent loss in cauliflower has been reported.

Symptoms: The poor emergence of seed and sudden falling off the seedlings are the major symptoms of damping off. This nursery disease is favored by pathogen inoculum and favourable environmental conditions. The wire stem stage is different to damping off where light to dark brown areas appear on the stem extending 1-2 cm below the soil line.



Fig. 64: Pre emergence Damping off of cauliflower seedlings

The lesions girdle the stem, which become sunken and at later stage turn in to tough and woody structure. The stem area becomes wiry in appearance, relatively thinner than the healthy stem known as wire stem. The lower leaves loose turgidity and give wilting appearance and the affected plants die prematurely. Plants contracting infection at later stages give unthrifty, stunted appearance and produce small sized curds. The infection is prominent at the beginning of head formation, when the lower leaves develop dark brown, oval lesions. The lesions turn soft and watery due to secondary infection. The pathogen, *Rhizoctonia solani* with *Pythium* causes damping off in nursery, and after transplanting it inflicts wire stem disease that out rightly kills young seedlings in main field (Fig. 64).

Causal organism and key characteristics: The seedlings of cabbage and cauliflower suffer due to the attack of *Pythium* spp., *Fusarium* spp., and *Rhizoctonia solani* at nursery stage. The species reported by various workers are *Pythium ultimum* Trow. var. *ultimum*, *Pythium aphanidermatum* (Edson) Fitzpatrick. *Rhizoctonia solani* produces typical wire stem symptoms. This fungus produces brown hyphae that are up to 12 µm in diameter, has dolipore septa, with right angle branching and a crosswall in the adjacent branch. Since this fungus does not produce asexual spores, the perfect stage develops white, thin, hymenial layer near the collar region which comprises basidia and hyaline basidiospores that measure 6-14 x 4-8 µm. This disease is highly favored by the temperature in the range of 20 to 25°C and relative humidity from 95 to 100%. The fungus is polyphagous in nature and has been recorded on several hosts from various parts of the world. It attacks 287 genera belonging to dicotyledonous and monocotyledonous families of Angiosperms and three each of Gymnosperms and Pteridophytes.

Disease cycle and favorable conditions: The soil borne nature of *Pythium* spp. allows the pathogen to survive in soil or plant debris for prolonged periods. The seedlings get affected before emergence lead to poor germination. Post emergence the seedlings topple down or die are said to "damp-off." The severity of the disease depends on the amount of pathogen in the soil and environmental conditions. Cool, cloudy weather, high humidity, wet soils, compacted soil, and overcrowding especially favor development of damping-off. Fields with unequal levels of nitrogen, phosphorus and potassium, soil pH ranging between 7.4 and 8.5 having sandy-silt loam texture predispose the plants to the attack by pathogen. Application of ammoniacal form of nitrogen decreases the disease incidence as compared to nitrate form. The seedling stage upto 20 days is more prone to attack of *R. solanica* compared to 35 days and 50 days old plants. Sandy soil favors maximum wire stem incidence while clay soil the least. The fungus hibernates and perpetuates in the soil and when seedlings topple down and perish. The primary source of inoculum of *R. solani* is in the form of mycelium or sclerotia favoured by high humidity and temperature. The sclerotia are resistant to drought and cope large fluctuations in temperature. It is present in most soils and once established, remains there indefinitely. It spreads through rain, irrigation water, agricultural tools etc.

Disease management

Cultural practices:

Use of healthy seed, change of nursery site every year, disinfection of the plant bed either with soil solarization or by fumigants like formaldehyde, deep ploughing during summer months, shallow seeding to encourage rapid emergence and selection of the healthy seedlings at the time of transplanting should be followed to keep the disease under check. The disease reduced appreciably by treating the nursery soil with powders of commercial cellulose, rice stubbles or water hyacinth biomass in combination with NH_4NO_3 is very effective. Total microbial population also increased in amended soil. It is observed that when C: N ratio increase in soil, bacterial and actinomycetes increase and the contrary fungal population decreases.

Biological control:

Applications of bioagents either as seed treatment or soil application have been competent to manage the disease incidence. A spore mixture of *Streptomyces arena* and *S. chibaensis* was found to be effective against the disease besides improving shoot length and dry mass of seedlings. Seed treatment and soil application of *Trichoderma* suppressed damping off symptoms and increased the growth of plants (Roy et al., 1998). *Pseudomonas* strains release antibiotic and produce siderophore and therefore, seed treatment helps in improving germination and growth of seedlings, yield and chlorophyll has also been observed by seed and soil application of *T. harzianum*.

Integrated management:

The nursery from the ground level should be raised to 10 cm. above to help good drainage. Soil solarization of nursery soils for 2-3 weeks by using 45micron thick polythene sheet helps in suppression of pathogen population. Treatment of nursery soil with 10-15 g effective strain of *Trichoderma* along neem cake at 50 g / m² impregnated is very effective in raising healthy nursery. Seed treatment with *Trichoderma* @ 4 g / kg, seedling dip for 30 min with *Trichoderma* @ 10 g / litre helps to manage rots in the nursery. Soil drenching with captan 75 WP @ 2.5 gm/ litre, adoption of wide spacing of 60 x 50 cm, growing of Indian mustard as trap crop after every 25 rows of cabbage (One row of mustard sown 15 days before and second 25 days after planting of cabbage), first and last row should be of mustard are recommended to develop damping off free nursery.

Name of disease: DOWNTY MILDEW

Geographical origin, distribution and crop losses: The disease has several phases and commonly cause severe damage to seedlings in the nursery. It also causes extensive damage to the foliage at post transplanting stage in cabbage and cauliflower. It is a serious threat to the well-established cauliflower seed industry (Mahajan et al., 1995). Downy mildew has worldwide distribution. It was first recorded in 1883 at USA on *Brassica* spp. (Farlow, 1883) and subsequently from other parts of the world (Channon, 1981). In India, this disease was first reported by Butler (1918). The loss is enormous when it occurs in nursery, where it kills large numbers of plants and retard growth of others. The disease is serious during both rainy and winter seasons and about 75-90% seedling mortality has been recorded in Pune, Maharashtra (Gaikwad et al., 2004).

Symptoms: Important cole crops grown in India is cauliflower and cabbage. Downy mildew is serious and may appear from nursery to curd formation stage. Symptoms are observed on the leaves. Fine hair like downy growth of fungus is observed on the lower surface of leaves (Fig. 65). Corresponding to the fungal growth there is minute pinhead brown necrotic spots visible on the upper surface of leaves, which later coalesce to each other.



Fig. 65. Symptoms of downy mildew on cole crops

Causal organism: Downy mildew is caused by *Peronospora parasitica*.

Key characteristics: It is an obligate pathogen and forms mycelium and haustoria inside host tissues. Sporangia emerge through the stomata and are dichotomously branched and terminate with slender tips. Asexual spores are ephemeral and therefore rapid dispersal and infection is essential.

Disease cycle and favorable conditions: The fungus survives between crop season as oospores or on cruciferous crop and weed plants. It also survives as sporangia on leaves and inflorescence and as latent systemic mycelium in seeds or infected plant debris (Saharan et al., 1997). Infection is favoured by low temperatures and high atmospheric humidity following rain or dew. The penetration is usually direct but occasionally occurs through

stomata. Primary infection occurs due to soil borne oospores while sporangia released from sporangiophores found on the cotyledons or hypocotyls favour secondary spread. The fungus produces an abundance of sporangia that can be disseminated by wind and rain splash. Water droplets also help pathogen dispersal over short distances. *P. parasitica* on cabbage can be very destructive at 10-15°C, and symptoms can develop at 24°C, but sporulation is limited beyond 24°C.

Management

- Field sanitation, disease free seed and crop rotation reduce pathogen inoculum.
- Foliar spray of zineb 75%WP @0.2% at disease initiation stage and repeat next spray at 7 days interval.
- One spray of azoxystrobin 4.8% w/w + chlorothalonil 40% w/w SC 0.3%.
- Use sticker @0.1% with fungicide to avoid runoff of droplets.

Name of disease: ALTERNARIA LEAF SPOT

Geographical origin, distribution and crop losses: *A. brassicae* was first reported in 1909 from U.S.A. on cabbage while *A. brassicicola* on cabbage in 1924 from Holland. In India, it is noticed in areas where moderate temperature and humidity prevails during crop season. *A. brassicae* is encountered more frequently than *A. brassicicola* in different cauliflower cultivars in Haryana and Sikkim. *A. brassicae* and *A. alternata* were reported on cauliflower from Hooghly district of West Bengal. Alternaria affects cauliflower at all stages of growth causing 80 per cent reduction in seed yield and rotting of curds making the produce commercially unmarketable.



Fig. 66. Symptoms of Alternaria blight on siliqua

Symptoms: Alternaria leaf spot is usually appeared in early stage of plant growth in cauliflower while in later stage in cabbage. Alternaria leaf spots are restricted to lower leaves only and do not cause economic loss to the crop except when some of the hybrid varieties of cauliflower are used. Symptoms appear as circular light brown spots on leaves. Concentric rings are clearly visible on the spots. Black sporulation is observed in humid weather. Cabbage infections do not extend very far in the host tissue. Inflorescence and siliqua are severely infected during seed crop (Fig. 66). The curd of cauliflower infected as brown discoloration of individual florets and flower clusters.

Causal organism: The disease is caused by *Alternaria brassicae* and *A. brassicicola*.

Key characteristics: *Alternaria* spp. are readily distinguished by spore size and morphology. *A. brassicae* conidia are obclavate with one slender, unbranched beak that measures 55-75µm long; beaks extend from the apical end of the spore body. Conidia have six to 19 transverse septa and one to eight longitudinal or oblique septa and are borne in short chains of up to four spores. Both *Alternaria* species require free water for infection. Sporulation of these pathogens is favored by alternating light and dark periods. Spores are dispersed by splashing water and by wind. *A. brassicicola* germinated readily and required lesser incubation periods.

Disease cycle and favorable conditions: *Alternaria* spp. perenniates as spores or mycelium on infected plant residues or on infected seeds and cruciferous weeds. When seed borne, pathogen may directly attack seedlings and cause damping off, stem lesions or collar rot. Most often fungus sporulates on plant residues during periods of rain, heavy dew, or conditions of high soil moisture and dispersed locally by rain splashes and wind currents. The pathogen is known to sporulate abundantly in decaying vegetable material in the soil. These two

pathogens also affect many crucifer weeds and may serve as alternate hosts. Insect transmission also has been reported for *A. brassicicola*.

Management

- Alternaria leaf spot of cole crops is effectively managed by detaching all the infected lower leaves in morning and then burning.
- Foliar spray of zineb 75% WP @ 0.2% at disease initiation stage on leaf and second spray of chlorothalonil 75% WP. @ 0.2% along with sticker during siliqua formation.
- Use disease-free and certified seeds from healthy crop.

Name of disease: WHITE ROT

Geographical origin, distribution and crop losses: This disease is an important plant pathogen for its worldwide distribution, wide host range.

Symptoms: This disease is more common in snowball group of cauliflower in seed production of early cauliflower and cabbage. Symptoms appear as water-soaked rotting of curd, petiole, stalk and stump region of the leaves. Soon after infection, growth of white mycelium is observed all over the infected portion. Cauliflower grown for seed production is severely affected by the disease and entire inflorescence collapsed (Fig. 67). This mycelium develops honeydew stage after colonization of the tissue. Later, entire rotted portion is converted into compact mycelial mat followed by hard black sclerotial body. This is the resting structure and primary inoculum source.



Fig. 67. White rot symptoms on cauliflower

Causal organism: *Sclerotinia sclerotiorum*

Key characteristics: Ascospores of *Sclerotinia* spp. infect upper portions of plants to cause diseases such as flower blights, stem rots, fruit rots, and head blight. Initial water-soaked spots expand irregularly and indeterminately, and girdle affected stem, which causes distal portions of the plant to wilt and then become necrotic. Affected tissues may develop a soft watery and a thick white mycelium forms, often both inside and outside the tissue. As the disease progresses, desiccated plant tissues appear bleached compared to those senescing normally, and frequently, diseased tissue develops a shredded appearance.

Disease cycle and favorable conditions: Soilborne sclerotia of *S. sclerotiorum* most frequently occurs after a significant rain or irrigation event, and is aided by a shaded, slow-drying soil surface. Ascospores are forcibly discharged and extended periods of leaf wetness (16 to 48 hours) with temperatures in the range of 12 to 24°C promote ascospore germination and infection. Colonized 'weak' tissues then serve as a food base to grow and with adequate wetness, mycelium moves from the colonized tissue into the vigorous host tissues of stems, leaves, pods, etc. Later in the disease process, sclerotia form, either on the plant surface or within stems and other plant parts. As the plant or plant part dies, the sclerotia fall to the soil where they can survive for multiple years.

Management

- Main crop of cauliflower and cabbage should be periodically observed near the stump region in cool, cloudy and moist weather for primary infection.
- Cut the infected curds, leaves along with some healthy portion in morning and carefully collect in polythene to avoid falling of sclerotia in the field. Burn all these materials away from field.

- Foliar spray of mancozeb @0.25% along with sticker @0.1% at flowering stage in cool, cloudy and wet weather.
- Spray must cover stump and lower region of leaves.

Name of disease: BLACK ROT

Geographical origin, distribution and crop losses: Black rot of cole crops caused by *Xanthomonas campestris* pv. *campestris* (Pammel) Dowson is a very destructive disease, which significantly (> 50%) reduces the yield of crops all over the world under favorable environmental conditions.

Symptoms: First sign of the disease often appears along the margins of leaves as chlorotic lesions and chlorosis progresses in the direction of midrib usually forming V-shape area, which is the most characteristic symptom of the disease (Fig. 68a). Some of the veins and veinlets within chlorotic area turn black in severe case. Black vascular scar observed on any detached infected leaves, midribs and veins. Yellowing of leaves was observed from lower portion of mid vein in severe infection. Disease severity increases rapidly whenever hailstorm is taken place in cropping season. Symptom may appear from any side and center of the leaves (Fig. 68b). Severe infection of black rot is resulting complete burning of crop in cauliflower before the curb formation. The bacterium is transmitted through seed. The bacteria usually enter the cotyledons through stomata and pass to the young leaves and progress systematically throughout the plant system. The foliage infection and transmission are through water pores, insect injury, infested soil, storm, cultural practices and seedlings.



Fig. 68 (a-b). Characteristic 'V' shaped chlorosis complete burning of leaves due to black rot

Causal organism: The disease is caused by the bacterium *Xanthomonas campestris* pv. *campestris*.

Key characteristics: Nine races of Xcc have been identified based on the interaction between differentials with R genes and avirulence genes of the bacterial pathogen across the world. Races 1 and 4 predominate worldwide, whereas races 2, 3 and 5 are rare, and race 6 has been reported only from *B. rapa*.

Management

- Always collect seeds from disease-free plants for use in next year use.
- Certified seed should be preferred to minimum risk of seed inoculum.
- Crop rotation with non-cruciferous crops.
- Use intercrop of creeping type urd and mung as mulches to reduce rain splash.
- Detach the lower infected leaves in afternoon when dew and bacterial ooze dried up from the leaves and then burn it.

- Use antagonistic bacteria in the soil.
- Spraying of copper oxychloride @0.3% with sticker @0.1% should be used if Alternaria infection also exists on the foliage giving good control of both the diseases.

Name of disease: PHYTOPLASMA ASSOCIATED DISEASES

Brassica oleracea L.

Symptoms: Little leaf, phyllody, flat stem and shoot proliferation (Fig. 69)



Fig. 69. Little leaf, flat stem and shoot proliferation in cauliflower (Courtesy: Dr A. Sajeena, Kerala Agriculture, Univ, Kerala)

Distribution: New Delhi; Trivendrum, Kerala

Causal organism: *Ca. P. aurantifolia* strain (16SrII group) (Kumar et al. 2021); *Ca. P. cynodontis* (16SrXIV-A subgroup) (Sajeena et al. 2021)

Disease cycle and Epidemiology: These phytoplasma strains are very common in many plants species and grasses all over India and hence a major epidemiological significance and can cause losses to other crops through leaf hopper and plant hoppers (Rao 2021).

Onion and Garlic

Introduction

Onion (*Allium cepa* L.) is a major bulbous crop among the cultivated vegetable crops and is of global importance. About 175 countries grow onion. Leading onion-producing countries are China, India, United States, Turkey and Pakistan. India ranks second in area and production. Among the States, the area and production of onion is the highest in Maharashtra accounting 28 percent of the country's production followed by Gujarat, Orissa and Uttar Pradesh. Garlic (*Allium sativum* L.) belong to Alliaceae family, has been widely recognized as a valuable vegetable, spice and a popular remedy for various ailments. The principal medicinal uses of garlic is to prevent and treat cardiovascular disease by lowering blood pressure and cholesterol, as an antimicrobial, and as a preventive agent for cancer. The active constituents are several complex sulfur-containing compounds that are rapidly absorbed, transformed and metabolized. It is the second most widely cultivated *Allium* spp. Garlic has played an important dietary, as well as medicinal, role for centuries. Even today the medicinal value of garlic is widespread and fast growing. Among 140 growing countries, China ranks number one in production followed by India. The low productivity of garlic in India can be mainly attributed to the non-availability of virus-free planting material, which is of utmost importance in a vegetative propagated crop like garlic.

Onion and garlic are susceptible to numerous foliar, bulb and root pathogens that reduce yield and quality. Excessive rains, humidity, temperature, pests and disease are critical factors of risk to onion cultivation. In addition to adverse weather, during post-harvest period, lack of dormancy in bulbs, pest and disease infestation, leads to excessive storage losses in onion and is a major concern of economic viability. About >40 diseases caused by fungi, bacteria, viruses and nematodes are reported across the globe in these crops. The major diseases are being purple blotch (*Alternaria porri*), *Fusarium* basal rot and *Sclerotium* white rot and *Colletotrichum* neck rot and smudge are other important diseases of onion, causing bulb rot, during bulb formation stage as well as during storage. Besides these major pathogens, sometimes diseases caused by *Stemphylium vesicarium*, *Phytophthora* spp., *Pythium* spp., *Botrytis* spp. and *Peronospora destructor* causing considerable yield losses, sometimes to the extent of 100% crop losses.

Name of disease: PURPLE BLOTCH & STEMPHYLIUM BLIGHT

Geographical origin, distribution and crop losses: Purple blotch is one of the major foliar disease of onion and garlic which significantly reduces the yield of the crop and cause heavy economic losses to growers. Purple blotch disease is spread throughout the world and reduces yield up to onion bulb 97% (Kareem *et al.* 2012).

Symptoms: Purple blotch is a common disease of onion and garlic. All above ground parts of the plant is severely affected. The blotch usually appears on the oldest leaves. Thrips are pre-disposing factor for the pathogen infection. In the beginning, they are small, elongated sunken and whitish yellow with a purple centre (Fig. 70a). Under favorable conditions, these blotches enlarge, coalesce and covered with the conidia of the

fungus. Finally, the leaves dry, wither and fall. Flowering stem of onion seed crop is severely infected and collapsed. The bulb can also be affected at harvest when the fungus infection occurred at the neck, causing storage rot. The fungus over wintering on the residues of infected leaves. The disease is also seed borne. Another disease is also associated with the crop and caused by *Stemphyllium vesicarium*. Two pathogens are responsible for 30-40% yield loss in seed production. This disease is one of the most important foliar diseases in the northern parts of India. The infection occurs on the leaves in the form of small yellow to pale orange streaks in the middle of the leaves. These soon develop into elongate spindle ovate spots, which later turn brown in colour and coalesce (Fig. 70b). Leaves dry premature and affect bulb development.



Fig. 70 (a & b): Symptoms of purple blotch and stemphyllium blight

Causal organism: This disease is caused by *Alternaria porri*.

Key characteristics: *Alternaria porri* is belonged to class dothideomycetes, order pleosporales and is grouped in the pleosporaceae family. Plant pathogenic *Alternaria* fungi secrete toxins as secondary metabolites which led to the development of disease in the host plant by necrosis. Mycelium of *A. porri* is branched, coloured and septate. Conidiophores develop singly or in groups, septate. Conidiophores are solitary or found in groups and are purple in the young stage and become brown when old. Conidia arise singly, they are straight or curved and are pale to brown. They have several transverse and longitudinal septa.

Disease cycle and favorable conditions: This disease is more common in kharif season. The temperature of 28-30°C and 80-90% relative humidity is congenial for disease occurrence. Small, sunken, whitish flecks with purple colored centers are common symptoms occurring on leaves and flower stalks. Further, large purple area develops forming dead patches. The intensity of disease varies from season-to-season. It causes losses of 25% in rabi, 50% in kharif in Maharashtra, while in Northern and Eastern parts 25-90% damage in bulbs and seed crop occurs when the disease appears along with stemphyllium blight.

Management

- Use disease free and certified seed.
- Crop rotation is recommended with non-host crop.
- Summer ploughings reduce the disease severity.
- Fungicidal spray of tebuconazole 25% WG @ 0.03% alternated by azoxystrobin 18.2% + difenoconazole 11.4% w/w SC @ 0.1% with sticker @0.1%.

Name of disease: Anthracnose/Twister

Geographical origin, distribution and crop losses: This disease was first recorded during 1924-1925 in the eastern and northern part of Puerto Rico (Nolla, 1926).

Symptoms: The initial symptoms of the disease can be seen as small white sunken spots which will later grow in size to cover the large area of the leaf. Apart from the lesion on leaves, there is abnormal elongation and twisting of pseudostem i.e. neck of the bulb. In the advanced stages of the disease, plants bent from the pseudostem. Seriously affected leaves can show the orange colours pigmentation of the fungus on the leaf. The progression of disease depends upon the frequency and intensity of rainfall. It has been found that the intensity of disease is higher in heavy soil than in light soil. In some cases, the disease is associated with the secondary infection of saprophytes in the bulb which give rise to rotting affecting the quality of the bulb (**Fig 71**).

Causal organism: *Colletotrichum coccodes*, *Colletotrichum circinans*, *Colletotrichum gloeosporioides*.



Fig. 71. Symptoms of Anthracnose

Disease cycle and favorable conditions: The fungus survives in plant debris and transmits through seedlings and onion bulbs. The pathogen normally infects plants from inoculum present in the soil. Conidia germinate and infect onion tissues under high humidity and temperatures of 23 to 30°C. Disease foci can be seen in patches that may result from soil-borne inoculums. The progression of Anthracnose depends upon the intensity and frequency of rainfall. Dispersal of conidia occurs from soil to lower leaves to the neck of the onion bulb by rain splashes.

Management: Combined application of Captan and Paclobutrazol as protective/preventive spray application, the least disease incidence was recorded. However, plants applied with Carbendazim and Paclobutrazol have been reported least disease severity on both protective and curative spray application. The protective spray application recorded with lower disease incidence, severity, short neck and high yield than the curative spray applications.

Leafy and Rare Vegetables

Name of disease: Leaf Spots

Symptoms: This is a common problem of all the leafy vegetables. Symptoms appear as light brown, circular spots, surrounded by red margin (Fig. 72 a&b). The red circular halo is not prominent in many vegetables except spinach. Disease is caused by *Cercospora* sp. Lettuce, celery and Chinese cabbage are affected by *Alternaria* sp. Symptoms are light brown, and circular spots on all over leaf lamina.

Causal organism: *Cercospora* sp. and *Alternaria* sp.

Management

- Avoid dense sowing of spinach.
- Collect lower leaves and burn it.

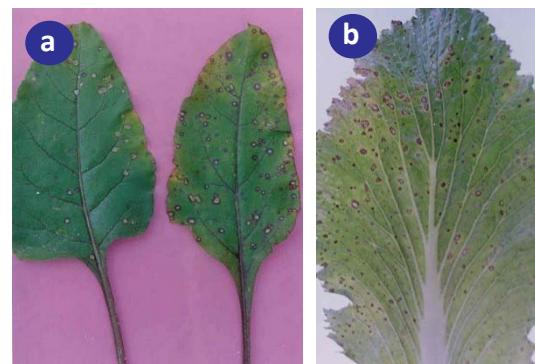


Fig. 72 (a &b) Symptoms of Cercospora leaf spots on spinach

Name of disease: WHITE ROT

Geographical origin, distribution and crop losses: This disease is an important plant pathogen for its worldwide distribution, wide host range.

Symptoms: This disease is caused by ubiquitous soil pathogen *Sclerotinia sclerotiorum*. It is more common in cool and humid weather. Symptoms appear as water-soaked rotting of petiole, stalk, stump region and cover portion of the leaves. Soon after infection, white mycelium is observed all over the infected portion. This mycelium develops honeydew stage after colonization of the tissue. Later on entire rotted lower portion and stump is converted into compact mycelial mat followed by hard black sclerotial body (Fig. 73). This is the resting structure and source of primary inoculum.

Causal organism: *Sclerotinia sclerotiorum*



Fig. 73: White rot infected cabbage with sclerotial bodies

Key characteristics: Ascospores of *Sclerotinia* spp. infect upper portions of plants to cause diseases such as flower blights, stem rots, fruit rots, and head blight. Initial water-soaked spots expand irregularly and indeterminately, and girdle affected stem, which causes distal portions of the plant to wilt and then become necrotic. Affected tissues may develop a soft

watery and a thick white mycelium forms, often both inside and outside the tissue. As the disease progresses, desiccated plant tissues appear bleached compared to those senescing normally, and frequently, diseased tissue develops a shredded appearance.

Disease cycle and favorable conditions: Soilborne sclerotia of *S. sclerotiorum* most frequently occurs after a significant rain or irrigation event, and is aided by a shaded, slow-drying soil surface. Ascospores are forcibly discharged and extended periods of leaf wetness (16 to 48 hours) with temperatures in the range of 12 to 24°C promote ascospore germination and infection. Colonized 'weak' tissues then serve as a food base to grow and with adequate wetness, mycelium moves from the colonized tissue into the vigorous host tissues of stems, leaves, pods, etc. Later in the disease process, sclerotia form, either on the plant surface or within stems and other plant parts. As the plant or plant part dies, the sclerotia fall to the soil where they can survive for multiple years.

Management

- Cut the infected curds, leaves along with some healthy portion in morning and carefully collect in polythene to avoid falling of sclerotia in the field. Burn all these materials away from field.
- Main crop should be periodically observed near the stump region in cool, cloudy and moist weather for primary infection and soon uproot it.
- Avoid dense planting and thick canopy.

Name of disease: *PHYTOPLASMA ASSOCIATED DISEASES WITH LACTUCA SATIVA L.*

Symptoms: Yellow leaf, Flat stem and malformation (Fig. 74)

Causal organism: Peanut witches' broom (16SrII-B) (Arocha et al. 2008); *Ca. P. trifloii* (16SrVI-D subgroup) (Gopala et al. 2018)

Disease cycle and Epidemiology: Not known



Fig. 74: Flat stem and leaf malformation in lettuce at Delhi

Radish

Name of disease: ALTERNARIA BLIGHT

Geographical origin, distribution and crop losses: Alternaria blight caused by *Alternaria brassicae* and *A. brassicicola* are the most devastating ones which cause heavy yield losses and major constraint for high quality radish seed production. About 46.38% seed yield is lost due to this disease.

Symptoms: The disease is prevalent during January to March. All foliar part is affected by the pathogen. Symptoms appear as small circular dark, black spots on the leaves while irregular, oval to elongated lesions on siliqua and inflorescence (Fig. 75a). Infected seeds also cause pre and post emergence seed rot after sowing. Pericarp of seed is severely infected by the pathogen (Fig. 75b). Cotyledons of seed also infected resulting internally seed borne nature of the pathogen. Infected seeds act as a primary source of infection.



Fig. 75 (a & b): Symptoms of Alternaria blight on leaf and siliqua

Causal organism: Leaf spot caused by *Alternaria raphani* is serious disease of radish

Key characteristics:

Management

- Collect disease-free seed from healthy crop.
- Use disease-free and certified seed.
- Pluck the infected lower leaves from main root crops in February and burn it.

Carrot

Name of disease: WHITE ROT

Geographical origin, distribution and crop losses: The damage caused by the disease is particularly important in temperate regions where carrots undergo long-term storage.

Symptoms: It is more common in cool and humid weather. Symptoms appear as water-soaked rotting of stalk, stump and collar region engulfing lower portion of the leaves. This pathogen was first time reported from India to cause collar and root rotting symptoms in seed production field of the institute in Asiatic carrot. Soon after infection, white mycelium growth is observed all over the infected portion (Fig. 76a). This mycelium is converted into compact mycelial mat. Later on, entire infected crown portion of carrot including some of the edible root converted into black, hard and large sclerotium (Fig. 76b). Sclerotia are the source of primary inoculum and cause primary infection on flowers.

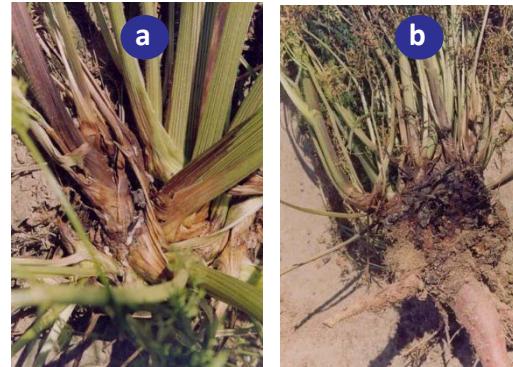


Fig. 76 (a&b): Water soaked rotten stalk of carrot with white mycelium

Causal Organism: This disease is caused by ubiquitous soil pathogen *Sclerotinia sclerotiorum*.

Key characteristics: Ascospores released by apothecia are the primary inoculum for diseases incited by *S. sclerotiorum* in the aerial parts of most hosts. In carrot crops, both mycelia and ascospores were considered important in initiating preharvest epidemics of stalk rot.

Disease cycle and favorable conditions: In field primary inocula for infection are airborne ascospores and soil borne hyphae. Infective mycelium arising from lesions on foliage can progress through the petiole toward the crown, from which it enters the root. Following the crown pathway, mycelium can circumvent the periderm, which may be a structural barrier to penetration from the exterior of the root. Optimum conditions for infection of carrots are prolonged periods of high moisture and temperatures of 13 to 18°C.

Management

- Uproot the infected carrot plant in morning carefully in polythene to avoid falling of sclerotia in the field. Burn all these materials away from field.
- Seed crop should be periodically observed near the stump and collar region in cool, cloudy and moist weather for primary infection

Name of disease: COLLAR ROT

Geographical origin, distribution and crop losses:

Symptoms: The disease is caused by *Sclerotium rolfsii* and is becoming ubiquitous with broad host range. Initially wet and soft rotting of carrot root is observed. Rotting covers entire root along with stump and leaves of the plant near collar region. Characteristics symptoms observed as white, cottony, fungus growth on root (Fig. 77). Gradually this hyphal mat is converted into small, mustard like sclerotia that survive in the soil.



Fig. 77. Symptoms of collar rot on carrot

Causal organism: *Sclerotium rolfsii*

Management

- Crop rotation with low land paddy and other cereals.
- Field sanitation by uprooting and burning of all infected plants.
- Summer fallowing, ploughing accompanied with one irrigation followed by again ploughing in summer to reduce the soil inoculum.
- Green manuring in June and July followed by soil application of *Trichoderma* @5 kg/ha soon after ploughing of the sun hemp.
- Seed treatment with *Trichoderma* @ 0.6 to 1% depending upon soil and pathogen status.
- Remove all the weeds from field.

Name of disease: CARROT PHYLLODY & WITCHES' BROOM

Symptoms: Witches' broom and violet leaves (Fig. 78).

Distribution: Delhi, Gorakhpur (Uttar Pradesh), Pantnagar (Uttarakhand)

Causal organism: *Ca. P. asteris* and *Ca. P. australasia* related strains (16Srl-B/16Srl-C) (Arocha et al. 2008; Dinesh et al. 2021)

Disease cycle and Epidemiology: Several weeds are known to host of these phytoplasma strains all over India and vectors are known (Rao 2021)



Fig. 78: Symptoms recorded in carrot: a. Witches' broom and phyllody at Experimental field, Division of Vegetable Science, IARI, New Delhi. b. Carrot leaf purpling and stunting symptoms at Kushmi Jungle, Eastern Gorakhpur, Uttar Pradesh.

Elephant Foot Yam

Introduction

Elephant foot yam (EFY), *Amorphophallus campanulatus* (syn. *A. paeoniifolius*), commonly known as 'zimikand', 'suran' or simply elephant foot, is one of the most nutritious tuberous vegetable crops. It is commonly grown in India, China, Africa, other South-East Asian and Pacific countries. It is a rich source of carbohydrates, minerals (Ca, P etc.) and Vitamin A and B. The calcium oxalate crystals present in the corms of wild species causes acridity and irritation and this can be overcome to a great extent by boiling. Apart from vegetables, this is also used for various medicinal purposes. In India, this crop is traditionally cultivated in West Bengal, Andhra Pradesh, Tamil Nadu, Bihar, Gujarat, Kerala, Uttar Pradesh and Jharkhand. These not only enrich the diet of people but also possess medicinal properties to cure many diseases. Several abiotic and biotic stresses play an important role in the production and productivity of the crops. Among the biotic stresses, diseases are the key factors which determine the successful production of these crops. Diseases can sometimes, be a limiting factor in the production of elephant foot yam. The crop is affected by many diseases such as collar rot (*Sclerotium rolfsii*), mosaic disease (*Dasheen mosaiv virus* (DsMV)), leaf blight (*Phytophthora colocasiae*), corm rot (*Rhizoctonia solani*), bacterial leaf blight (*Xanthomonas amorphophalli*), root knot nematode (*Meloidogyne incognita*) and lesion nematode (*Pratylenchus spp.*) in some Southern states of the country.

Name of disease: COLLAR ROT

Geographical origin, distribution and crop losses: Collar rot caused by fungus *Sclerotium rolfsii* is the most serious disease of EFY. The pathogen *S. rolfsii* is a destructive fungus affecting more than 500 plant species of about 115 families and includes several important vegetable and other crops. The soil borne sclerotia and wide host range of the fungus makes its control very difficult. The disease has been reported from almost all the EFY growing areas of India. The yield losses upto 100% has been reported due to this disease. Disease has been reported from all the states of India.

Symptoms: Crop is severely affected by disease with 32.9% incidence as complete killing of the plant. Initially yellowing of the plant is observed and later plant toppled down before maturity of the rhizome (Fig. 79). Close observation revealed wet rotting of apical portion of rhizome and base line stem. Rotting covers entire collar region of the rhizome that remain continue during storage also. Characteristics



Fig. 79: Yam crop infected with collar rot

symptoms observed as white, cottony, fungus growth and sclerotia on affected portion as well as on contact soil. Gradually this hyphal mat is converted into small, mustard like sclerotia that survive in the soil.

Causal organism: The disease is caused by *Sclerotium rolfsii* and is becoming ubiquitous with broad host range.

Key characteristics: It produces white, fluffy, branched, fine thread like mycelia which forms numerous mustard seed like small sclerotia. These sclerotia survive in soil, causing serious outbreaks of the disease in warm wet weather, following a significant dry spell. Pathogen can survive parasitically inside infected corms or saprophytically on plant debris or as sclerotia in the soil. When enough moisture is present, sclerotia germinate myceliogenic ally and infect the collar region of stem. The injury to the basal portion of stem due to intercultural operations and weeding facilitate severe infection by the fungus.

Management

- Remove all the sclerotia from apical portion of rhizome during planting.
- Crop rotation with low land paddy and other cereals.
- Field sanitation by uprooting and burning of all infected plants.
- Summer fallowing, ploughing accompanied with once irrigation followed by again ploughing in summer to reduce the soil inoculum.
- Green manuring in June and July followed by soil application of *Trichoderma* @5 kg/ha soon after ploughing of the sun hemp.
- Rhizome dipping in *Trichoderma* @1% solution for 20 minutes.
- Remove all the weeds from field.
- Drenching of *Trichoderma* @1% suspension after 25 and 40 days of sowing.

Potato

Introduction

Potato (*Solanum tuberosum* L.) ranks first as a non-cereal food crop in terms of global consumption. The capability of producing highest dry matter, energy, and edible protein per unit area per unit time makes potato a fastest growing food crop especially in developing countries and that's why it is regarded as the future crop for assuring food security. Globally potato occupies 19 million hectares area with a production of 388 million tones. China and India respectively are the leading potato producing countries in the world and together they contribute to 38% of global potato produce. In India, potato is grown on 2.13 million hectares with production and productivity of 53 million tonnes and 20.5 tonnes, respectively.

Name of disease: LATE BLIGHT

Geographical origin, distribution & losses: Late blight caused by *Phytophthora infestans* is considered as the most threatening biotic stress for food security. The pathogen is considered as re-emerging due to regular emergence of its novel strains with increased virulence and its appearance in new locations with surprising intensity. *Phytophthora infestans* originated in the Americas; both central Mexico and the South American Andes have been proposed as its centre of origin. The disease is widely distributed throughout the world but is more serious in temperate highlands. Losses due to late blight have been estimated to • 12 billion per annum of which the losses in developing countries are to the tune of • 10 billion per annum.

Symptoms: Late blight affects foliage, stems and tubers. It appears on leaves as water- soaked irregular pale green lesions mostly near tip and margins which rapidly grow into large brown to purplish black necrotic spots (Fig. 80a). A white mildew, which consists of sporangiophores and spores of the pathogen, can be seen on lower surface of the infected leaves especially around the edges of the necrotic lesions (Fig. 80b). In dry weather the water-soaked areas dry up and turn brown. On stems and petioles light to dark brown lesions encircle the stems as a result the affected stems and petioles become weak at such locations and may collapse. The pathogen can sporulate from stem lesions under very wet and moderate temperature conditions (Fig. 80c). Entire crop gives blackened blighted appearance (Fig. 80d) especially under disease favorable conditions and may be destroyed within a week. Tubers in soil become infected by rain borne sporangia coming from the diseased foliage and show irregular reddish brown to purplish areas which extend into internal tissues of the tubers (Fig. 80e). The infected tubers usually are hard, dry and firm but may get attacked by soft rot causing bacteria and rot in field and stores. Sepals, flowers and berries may also get infected and show white mildew under humid and cloudy weather.

Causal organism & key characteristics: Late blight, caused by the oomycete *Phytophthora infestans* (Mont.) de Bary is heterothallic and requires two mating types (A1 and A2) for sexual reproduction. The pathogen is characterized by lemon shaped detachable, papillate sporangia produced on sympodially branched

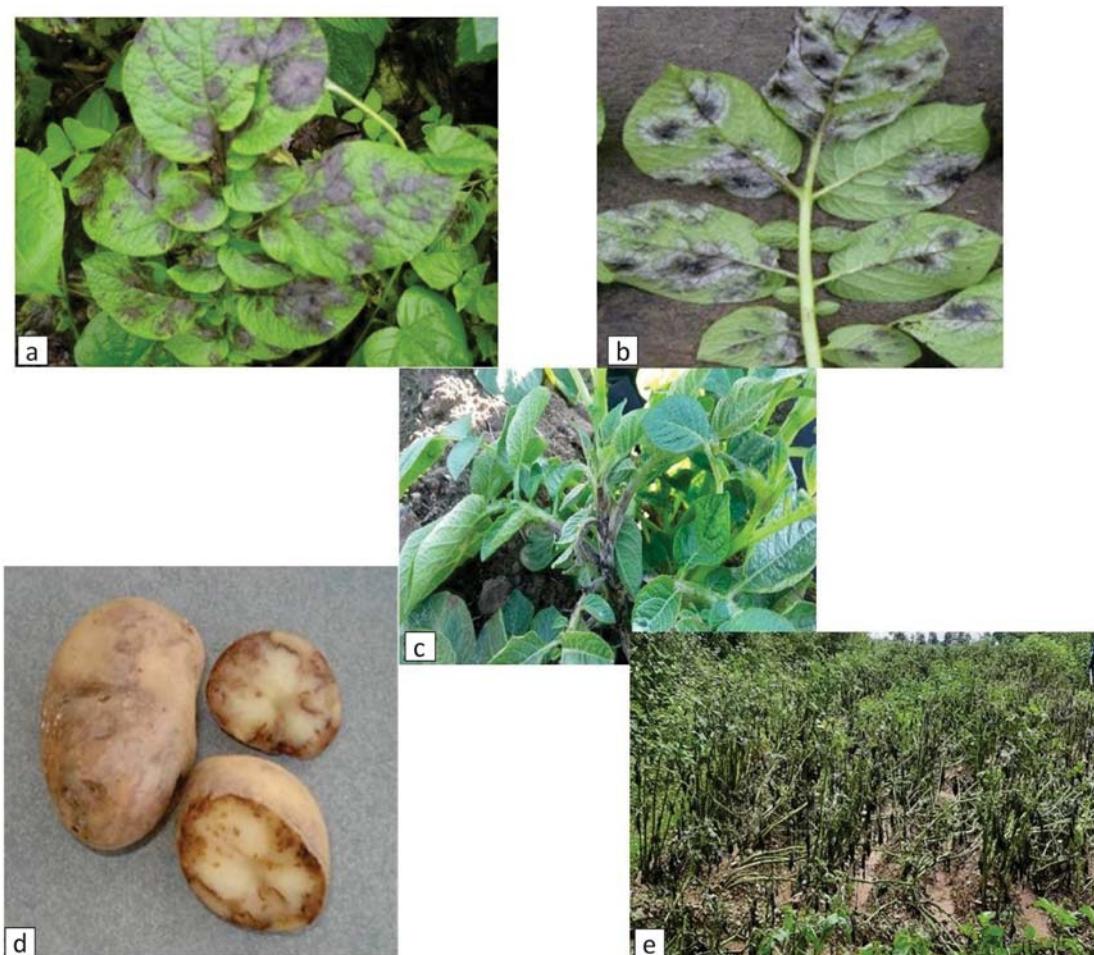


Fig. 80: (a-d) Late blight symptoms on various plant parts e) Late blight infected field

sporangia of indeterminate growth. The sporangiophores exhibit a characterized swelling at junction where sporangia are attached with the sporangiophores. Sexual reproduction results in production of thick-walled oospores that can survive adverse conditions. The vegetative cells are diploid but higher ploidy levels have also been described. The genome size of the *P. infestans* is considerably larger (240 Mb) having an extremely high repeat content (~74%), which is thought to contribute to *P. infestans* evolutionary potential by promoting genome plasticity, thus enhancing genetic variation of effector genes leading to host adaptation.

Disease cycle and favourable conditions: Both sexual and asexual reproductive cycles are operative; however, the asexual cycle predominates. Asexual cycle is the major contributor to the epidemics as many cycles in a season are rapidly produced. Sporangia are produced under conditions of high moisture and moderate temperature (10-24°C). Sporangia produced on leaf or stem lesions are dispersed by air currents or dislodged by raindrops. Once in the air, sporangia can survive several hours under cloudy conditions, but only up to an hour under sunny conditions. Sporangia germinate either directly, by forming a germ tube (at temperature >15°C), or indirectly, by producing zoospores (<15°C). Under optimal conditions (18-22°C), symptom development takes place within 3 days while in another 2-3 days; pathogen starts sporulation from the infection site. Moderate temperatures (10-25°C) and very wet conditions (humidity near saturation) are required for sporulation. The survival between cropping periods is often dependent on the survival of infected tubers. Thus, tuber infections are very important in the epidemiology of *P. infestans* in potatoes. In locations where A1 and A2 individuals coexist, hybrid oospores can be formed that can survive for months, perhaps years, in soil and can act as source of primary inoculum.

Management: Management of this devastating pathogen is challenged by its remarkable speed of adaptation, with respect to emergence of virulence towards resistant cultivars, and to fungicide resistance. No single approach is effective, hence combination of approaches in an integrated manner are essential to combat this devastating disease.

- ❖ Reducing the primary source of inoculum is the critical management strategy as onset of epidemic can be delayed by 3 to 6 weeks if all primary infection from early potato is eliminated. This can be achieved by eliminating volunteers and cull piles, waste heaps, infected tubers, use of certified seed and resistant varieties, balanced fertilization, adequate space between rows and plants, rotation with non-host crops, adequate hilling, harvest in dry conditions and when the tubers are mature. A sound crop rotation for 3 to 4 years is an effective way of reducing the risk of soil-borne inoculum as oospores can remain infectious up to 48 months in soil. As soon as the weather conditions become congenial for late blight, irrigation should be stopped wherever applicable. Only light irrigation may be given later, if required. High ridging is often used to reduce tuber contamination by blight. Another approach to reduce tuber blight is to destroy the canopy when blight reaches to 75% severity.
- ❖ Planting resistant cultivars can help to suppress late blight epidemics. With the use of host resistance, fungicide load can be reduced either by lowering the fungicide dose or increasing the application intervals.
- ❖ Fungicides with protectant and curative properties are registered for use against late blight on potato. Fungicides should be applied prophylactically to prevent germination and penetration of host tissues by the pathogen. Systemic or translaminar fungicides should be applied to cure the infection. Many forecasting models/decision support systems are available to predict the appearance of late blights which can be efficiently used to schedule fungicide applications.

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Name of disease: Early Blight

Geographical origin, distribution & losses: The first reference to the fungus as a parasite and its association with potato leaf blight goes back to 1891 when Galloway reported from Australia. Early blight has become widespread and turned into one of most important diseases after late blight. The disease occurs worldwide and is prevalent wherever potatoes are grown. Crop losses due to early blight during epidemics are estimated to the tune of 20-30% and annual expenditure globally on fungicides for control of *Alternaria* spp. is around \$45 million in potatoes.

Symptoms: Symptoms occur on foliage and tubers of potatoes. Infected leaves show small, round, oval or

angular, dark brown to black, dry and papery necrotic spots which have angular margins (**Fig 81a**). These spots are generally limited by leaf veins. The spots may or may not have concentric rings. The rings are more prominent in large blotchy spots that give them a bull's eye or target board appearance which are characteristic of the disease (**Fig. 81b**). As lesions expand and new lesions develop, entire leaves may turn chlorotic and dehisce leading to significant defoliation. Symptoms on potato tubers are characterized by sunken, irregular lesions which are often surrounded by a raised purple border. Beneath the surface of the lesion the tuber tissue is leathery or corky with a brown discoloration. Early blight lesions on tubers tend to be dry and are less prone to invasion by secondary organisms than lesions of other tuber rots. However, after prolonged storage severely diseased tubers may become shriveled.

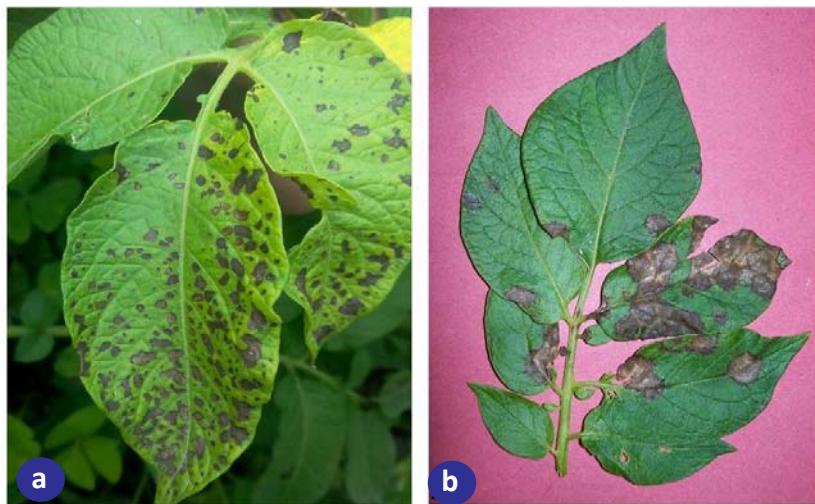


Fig. 81: Symptoms of early blight disease on leaves

Causal organism & key characteristics: In the past, potato diseases caused by *Alternaria* species were described as early blight (caused by *A. solani*) and brown spot (caused by *A. alternata*) disease. However, both diseases are often observed together on the same leaves and initial early blight symptoms are like brown spot symptoms, making them hard to discriminate. Recent studies have shown that the *Alternaria* population on potato is much more elaborate than a two-species disease complex. Several research groups have reported both small-spored species, such as *A. tenuissima*, *A. arborescens* and *A. infectoria*, and large-spored species, such as *A. grandis* and *A. tomatophila*, residing on potato leaves. Ten species of *Alternaria* have been implicated to cause foliar diseases of potato worldwide. These include *A. solani*, *A. alternata*, *A. tenuissima*, *A. dumosa*, *A. arborescens*, *A. infectoria*, *A. grandis*, *A. interrupta*, *A. longipes* and *A. arbusti*. The sexual stage of the fungus is not known. The asexual conidia are borne singly or in a chain on distinct conidiophores. Conidia are dark, ellipsoid to oblong with 9–11 transverse septa and tapered to a long beak. The fungus is readily cultured on artificial media where it produces a deeply pigmented gray/black hairy colony. The mycelium is haploid and septate, becoming darkly pigmented with age. Sporulation in culture can be stimulated by exposure to fluorescent light.

Disease cycle and favorable conditions: The pathogen overwinters primarily on infected crop debris. The dark pigmentation of the mycelium increases resistance to lysis which extends the survival time in the soil. Thick-walled chlamydospores have been reported, but they are found infrequently. In mild climates the pathogen can survive from season to season on volunteer potato plants as well as other weedy Solanaceous hosts such as horse nettle and nightshade. Warm, humid (24–29°C) environmental conditions are conducive to infection. Desiccated germ tubes are able to renew growth when re-wetted, and, hence, infection can occur under conditions of alternating wet and dry periods. Secondary spread of the disease results from conidia being dispersed mainly by wind and occasionally by splashing rain or overhead irrigation. Early blight is considered polycyclic with repeating cycles of new infection. Spores produced on foliar lesions also contaminate soil and infect tubers through wounds during harvest.

Management

- ❖ Follow sound cultural practices to remove infected crop debris, volunteer plants and weeds. Harvest tubers at optimal maturity and avoid injury to tubers. Avoid irrigation in cool, cloudy periods or late in evening when foliage may stay wet for extended periods. Maintenance of adequate soil fertility levels is also critical for managing early blight as the disease is often associated with crops suffering from a lack of nitrogen, particularly towards the end of the growing season on older senescing foliage.
- ❖ Several protectants (e.g. chlorothalonil, dithiocarbamates) and systemic (e.g. Quinone outside inhibitors-QoI and succinate dehydrogenase inhibitors- SDHIs) fungicides are available for effective management of early blight. The efficacy of these fungicides could be enhanced by scheduling their application in accordance to forecasting models.

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Rhizoctonia Canker and Black Scurf

Geographical origin, distribution & crop losses: Black scurf is perhaps the oldest known disease of potato with worldwide distribution. It is prevalent in most potato growing regions of the world including India, North-West, Central Volga-Vyatka, Ural regions of Russian Federation, West and East Siberia, Far East, Baltic countries, Belarus, woodlands of Ukraine, north and central regions of Kazakhstan and many other potato growing countries. In India, it has spread widely in most potato growing regions. The disease kills potato sprouts, causes damping off of seedlings, delay crop emergence and reduce crop stand. The tuber quality is also affected by the development of sclerotia on progeny tubers, thus their market value is reduced. Yield losses up to 35% primarily due to reduced crop stand have been reported.

Symptoms: The most common symptoms of the disease appear on tubers as black irregular lumpy encrustations of fungal sclerotia which stick to the surface of tubers (black scurf). Reddish brown lesions develop on stems and often girdle them. Partial or complete girdling of the stems results in resetting of plant tops, purple pigmentation, upward chlorosis or rolling of leaves. Formation of aerial tubers in axis of leaves due to interference with starch translocation, are also observed in the infected plants (**Fig. 82**). Other symptoms on tubers could be cracking, malformation, pitting and stem end necrosis. The sexual or perfect stage of the pathogen appears on stem just above soil line as whitish grey mat or mycelial felt. These mats are often



Fig. 82: a) Stem canker b) Formation of aerial tubers c) purple pigmentation and rosetting of top leaves d) black scurf symptoms due to *R. solani*

located above a lesion on the below ground portion of stem and are generally visible later in the growing season under favorable weather conditions.

Causal organism(s) & Key characteristics: The disease is caused by a fungal pathogen *Rhizoctonia solani* Kuhn (perfect basidial stage: *Thenatephorus cucumeris* (Frank) Donk). *R. solani* consists of genetically defined populations that are distinguished by anastomosis between hyphae of the isolates belonging to the same 'anastomosis group' (AG). Fourteen different anastomosis groups viz., AG-1 to AG-10, AG-Bl, AG-11, AG-12, and AG-13 have been recognized. Isolates of *R. solani* from potato mostly belong to anastomosis group 3 (AG-3). However, strains belonging to other anastomosis groups (AG-1, AG-2-1, AG-2-2, AG-4, AG-5, AG-7 and AG-9) have also been isolated from potato stems, stolons, roots and tubers, as well as from soils in which potatoes were grown. In Central Mexico, in addition to AG-3, isolates of AG-2, 4, -5, and -7 have been collected from potato plants and/or tubers from fields. In India, only AG-3 (PT) isolates of *R. solani* have been found responsible for black scurf and stem canker disease of potato. However, since pathogen is tuber-borne in nature, there is every possibility of introduction of other pathogenic AGs in India with import of seed/ ware potato.

Disease cycle and favorable conditions: The pathogen is both soil and seed borne but the disease spreads to new growing areas primarily through sclerotia-covered seed tubers. Soil-borne infection emerges later in the season since the fungus needs some time to grow into proximity with its potato host. Sclerotia of the pathogen germinate between 8 to 30°C and invade emerging sprouts or potato stems. Optimum temperature for germination of sclerotia is 23°C and for development of stem lesions is 18°C. Very high summer temperatures are not conducive for production of sclerotia and their survival. High soil temperature (28-32°C) and high soil moisture favor development of sclerotia. Sclerotial development on tubers is initiated depending on environmental conditions. Maximum development of sclerotia takes place in the period between dehaulming and harvest of the crop. Late harvested crops show more black scurf incidence. The host range of *R. solani* is very wide and the pathogen causes diseases of many economically important plants belonging to Solanaceae, Fabaceae, Asteraceae, Poaceae and Brassicaceae as well as other ornamental plants and forest trees.

Management

- ❖ The use of healthy seed tubers free from sclerotia, seed tuber treatment with 3% boric acid (dip for 30 minutes/ spray) before cold storage, planting in relatively dry and warm soil with shallow covering of seed tubers to achieve rapid crop emergence with less opportunity for the fungus to attack, two to four year crop rotation with cereals and legumes, soil solarization with transparent polyethylene mulching during hot summer months especially in Indian subtropical plains has been found very effective for control of the disease.
- ❖ Bio-control agents such as *Trichoderma viride*, *T. harzianum* and fungicides such as benomyl, thiabendazole, carboxin, pencycuron and azoxystrobin have been reported effective for control of the disease.
- ❖ Boric acid and pencycuron are the two chemicals that are frequently used by Indian farmers to control black scurf. Seed tubers treatment with boric acid is recommended before sprouting usually prior to cold storage whereas pencycuron applied to the sprouted tubers at planting achieved more than 97% disease control by penflufen (0.062, 0.083%) dip treatment of scurf infected tubers for 10 minutes.

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Name of disease: FUSARIUM WILT AND POTATO DRY ROT

Geographical origin, distribution & crop losses: The disease was first recorded in Martius in year 1842 and the fungus was named *Fusisporium* which later identified as *Fusarium oxysporum*. The disease causes greater loss in USA, China, Bangladesh, Tunisia, Great Britain, Iran, Australia and Poland. Dry rot affects the crop stand by preventing the formation of potato sprouts, resulting in losses of up to 25% and infection rates as high as 60% during storage. This disease causes post-harvest losses costing US producers US\$100–250 million annually. In India, an estimated loss of 6-13% has been observed in cold stores. The amount of inoculum in the soil determines the amount of yield loss, which can range from 25 to 60%.

Symptoms: The disease occurs in two phases i.e. in the field as wilt and in storage as dry rot. In the standing crop, wilt symptoms are comprised of common dropping of leaves followed by whole plant dying within a week. The uprooted plants can be observed for necrotic brown discoloration of vascular regions in the stem (**Fig. 83a**). The main plant sections that are immediately affected by dry rot disease are tubers. On tubers, symptoms appear as small brown lesions on surface (**Fig. 83b**) which subsequently enlarge, appear dark, sunken, and wrinkled producing white, pink, or blue pustules. In later stages a cavity often develops in the centre and white cottony, orange or violet mycelia growth can be observed on heavily infected tubers (**Fig. 83c**) kept in cold stores. Rotten tubers may shrivel and get mummified.



Fig. 83: a) Wilt Symptoms caused by *Fusarium* on collar region b-c) Dry rot symptoms on tubers

Causal organism(s) & Key characteristics: Around the world, 16 different *Fusarium* species viz. *F. oxysporum*, *F. sambucinum* (Syn. *F. sulphureum*), *F. solani* var. *coeruleum*, *F. avenaceum*, *F. culmorum*, *F. acuminatum*, *F. crookwellense*, *F. equiseti*, *F. graminearum*, *F. scirpi*, *F. semitectum*, *F. sporotrichioides*, *F. ciliatum*, *F. reticulatum*, *F. torulosum*, *F. verticillioides* cause potato dry rot. The pure culture of *Fusarium oxysporum* bears floccose, sparse to abundant white mycelia with a pale violet tinge as observed on potato dextrose agar (PDA) media; while *F. oxysporum* bears colonies of brick orange mycelia with lobed margin. Macroconidia appear straight to slight slender, apically tapered, minute falcations, mostly 3-5 septate. Microconidia and chlamydospores are

also evident under favorable conditions.

Disease cycle and favourable conditions: The pathogen is both soil and tuber borne. The pathogen makes entry through wounds produced during harvesting, handling, and shipping, and these diseased tubers cause dry rot in storage. Infected tubers and soils adhering to the surface of tubers are primary source of inoculum. Seed tubers are contaminated with chlamydospores which survive in the soil as resting spores. The infection depends upon the species involved as sporulation varies among species. The favourable temperature for mycelial growth of *F. sambucinum*, *F. graminearum*, *F. oxysporum*, *F. solani* and *F. culmorum* is 20–25 °C. However, for sporulation 25–30 °C temperature is required. *F. solani* var. *coeruleum* requires 25–30 °C and 20–25 °C for growth and sporulation respectively. Interestingly, the *Fusarium sambucinum* has been reported to grow even at 4°C which is detrimental for table purpose potatoes kept in cold stores. The processing varieties which are kept at 12 °C are more vulnerable to dry rot infection as compared to table purpose varieties.

Management

- ❖ Avoid bruising and wounding during harvest and post harvest handling and provide suitable wound healing conditions through curing which limit the entry and germination of fungal spores.
- ❖ Rotation with non-host crops, use of disease-free seed tubers, and storage in a cool environment can be followed to further reduce the disease incidence.
- ❖ Because respiration in stored potatoes generates excessive CO₂ and heat, which may favor the formation of adhering fungus spores, enough circulation of cool air is critical during storage.

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Name of disease: CHARCOAL ROT

Geographical origin, distribution & losses: Charcoal rot is an important disease in tropical and subtropical regions which affects a broad range of host plants, including potato (*Solanum tuberosum* L.). The disease is serious in the southern United States, Hawaii, central California, Maryland, Illinois, Mediterranean region and in warm areas of India and Peru. The disease reduces marketable quality of tubers and yield losses up to 88% have been reported.

Symptoms: The pathogen attacks growing potato plants and tubers both at harvest and storage. Affected plants exhibit yellowing and wilting of plants. Tubers can become infected before harvest and during storage, and tuber infection can result in the loss of an entire crop. Initial symptoms on tubers develop around the eyes, lenticels and stolon end as a dark light grey, soft, water soaked lesions (Fig. 84). Later, these lesions become filled with black mycelium and sclerotia. Soft rot may subsequently develop from secondary invaders.



Fig. 84: Charcoal rot symptoms on potato tubers

Causal organism & key characteristics: The causal agent of charcoal rot is *Macrophomina phaseolina* (Tassi) Goidanich (Syn.: *M. phaeoli* (Maubl.); Tel.: *Botryodiplodia solani tuberosi* Thiram). Sclerotia formed within roots, stems, tubers and leaves are black, smooth, hard 0.1 to 1.0 mm in diameter. Pycnidia may develop on leaves and stems depending upon the strain of the fungus. Conidia are single, hyaline and ellipsoid to obovoid.

Disease cycle and favourable conditions: The fungus has wide host range, both cultivated and wild, and persists on dead or dying plant tissues and survives the unfavourable periods by forming microsclerotia. Both soil and infected tubers serve as source of inoculum. The pathogen spreads through the infected seed tubers and through the infested soil carried along with the implement. Temperature around 30°C or above are very favourable for infection, the rot is slow at 20 to 25°C and stops at 10°C or below. Poor plant nutrition and wounds predispose the plants to charcoal rot. Fungal growth stops in tubers placed in cold stores, but it resumes the growth after cold storage.

Management

- ❖ Disease can be managed through planting early maturing cultivars, frequent irrigations to keep the soil temperature down and harvesting potato tubers before the soil temperature exceeds 28°C.
- ❖ Do not delay harvest once tubers have matured and avoid bruising and wounding during harvest and post-harvest handling.
- ❖ Rotation with non-host crops, use of seed from disease free area, and storage in a cool environment can be followed to further reduce the disease incidence.

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Name of disease: WART

Geographical origin, distribution & losses: The origin of wart pathogen is in the Andean mountains of Latin America, where it co-evolved with potato and subsequently distributed around the world with tubers. The disease occurs in limited areas on all continents except Australia. Quarantine measures restrict its distribution. Yield losses in the range of 50–100 % have been reported.

Symptoms: Early infection of young developing tubers results in distortions and sponginess and makes them unrecognizable. In older tubers, the eyes are infected and develop into warty, cauliflower-like protuberances (Fig. 85). Belowground galls appear at stem bases, stolon tips, and tuber eyes while above ground galls appear on the upper stem and leaf. The shape of galls is usually spherical but can be irregular. The galls above ground are colored green to brown while belowground warts are colored white to brown. At maturity wart tissue becomes colored black and leads to total tuber decay. Potato wart does not kill its host plant. Below ground symptoms may not be evident until harvest.



Fig. 85: Wart infected tubers

Causal organism & key characteristics: *Synchytrium endobioticum* (Schilberszky)

Percival is an obligate, holocarpic, endobiotic parasite that does not produce hyphae but sporangia, which contain motile zoospores. The fungus is obligate soil-borne and produces persistent resting (winter) sporangia (sori) with a long life span of more than 30 years. The sori are released into the soil from the decomposition of warts. Summer sporangia develop in infected potato tissue leading to secondary zoospore infections and they are thin-walled and short-lived. Resting sporangia are golden brown, thick walled and spheroidal. Numerous pathotypes of the fungus have been identified.

Disease cycle and favorable conditions: Resting sporangia germinate to release haploid zoospores which migrate in soil water to infect susceptible host tissues. Zoospores encyst in host tissues and develop into sori which release haploid zoospores into neighboring host cells thus resulting in secondary disease cycle. Host cells surrounding the infected ones are subject to hyperplasia and hypertrophy, resulting in the formation of warts. Some zoospores conjugate and form a zygote which develops into resting sporangia. These sporangia are released into soil after the decay of host tissues. Inoculum is spread primarily by the movement of contaminated soil adhering to tubers, equipment, and other carriers. Contaminated manure is also considered as means of inoculum dispersal. Resting sporangia are dormant structures which can remain viable in soil for more than 30 years. Water is required for the germination of summer and winter sporangia and for the dispersal of the zoospores. Temperatures of 12–24°C favour infection; however, cool summers with average temperature of < 18°C and annual precipitation of 700 mm are important for the development of the disease.

Management

- ❖ Follow strict quarantine legislation to exclude the pathogen entry into disease free areas. Once introduced into an area, the pathogen cannot be eradicated.
- ❖ Control of the disease is possible only by cultivation of wart immune varieties. No effective soil treatments are available and cannot be applied on large field scale.
- ❖ Diseased potatoes should not be used as seed and warded lumps and potato peelings should not be thrown in the field or in the manure pit but destroyed by burning.

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Name of disease: SILVER SCURF

Geographical origin, distribution & crop losses: Potato silver scurf is a consistent common problem for potato stakeholders across the globe. Although the first incidence of the disease was reported in the year 1970s, but the problem gained much consideration during 1990s due to severe losses in storage units of North America and Europe. The disease has a worldwide distribution and huge economic importance owing to its incidence in field and storage. There are reports of as high as 93% disease incidence. The lesion of this disease can cover 11-13 percent of the tuber's surface area, the most of any skin blemish disease.

Symptoms: Small, localized, light brown, circular spots with indistinct borders frequently enlarge to cover a considerable area of the tuber are the characteristic symptoms of the disease. Affected areas have a distinct silvery sheen, particularly if the surface is wet (Fig. 86). The quantity of conidia and conidiophores on the lesions gives them a silvery appearance. These lesions increase the permeability of tuber skin, causing weight loss and tuber shrinking. Extensive sporulation occurs in depressions and crevices on the tuber towards the stolon end and the eyes. The infection can be seen on dead, decaying, and drying seed tubers below ground even after they have been planted. The scurf lesions are more definite and frequently have a sooty appearance caused by conidiophores and conidia.

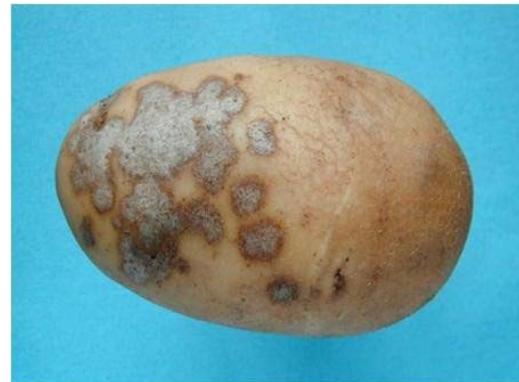


Fig. 86: Shiny and silvery lesions on infected tubers

Causal organism(s) & Key characteristics: *Helminthosporium solani* is the cause of silver scurf disease in potatoes. The fungus develops hyaline mycelia on artificial media, which appear brown septate, branched, and turns brown with age. Unbranched conidiophores are septate, with the conidia borne in whorls from the distal ends of the cells. Spores are 7-8 X 18-64 µm and have up to eight septa, and are dark brown, rounded at the base, and pointed at the ends. Septate conidiophores have a tiny apex and a broader base, and melanized conidia are produced in a basipetal sequence on them. On conidiophores, conidia have two to eight pseudosepta that create whorls.

Disease cycle and favorable conditions: *Helminthosporium solani* infects potato plants in the field and tubers during storage. The spread of the disease is through soil and seed, but seed potatoes are the original source of inoculum. The pathogen possesses polycyclic lifecycle and produces enormous inoculum. High humidity is necessary for disease development. The longer mature tubers remain in the soil, the more severe the problem becomes. Minimum conditions for infection are 3°C with 90% relative humidity; however, the optimum temperature for fungal proliferation is 20-25°C. Disease continues to increase in storage, and further infection may develop if the tubers are kept at high relative humidity and temperature. Sporulation is more abundant

on young lesions than on old ones. The pathogen is mainly tuber-borne but can overwinter in soil and may also survive between storage seasons inside potato storage, on potato waste and other organic substances.

Management

- ❖ Follow 3 years crop rotation with non-solanaceous crops (especially cruciferous crops) and harvest the tubers immediately after maturity.
- ❖ Rapid drying of tubers after harvest ensures suberization of wounds and longer curing decreases the incidence of the disease.
- ❖ Avoid failure of power during storage as it may lead to condensation events that result in more disease.

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Name of disease: POWDERY SCAB

Geographical origin, distribution & crop losses: Powdery scab was first intercepted in Germany in year 1841 and spread across Europe by 1855. The earliest record of this disease in India corresponds to the year 1921. The pathogen is widespread and occurs in most potato-producing areas of the world. It is primarily a disease of cool climates. Powdery scab of potato is currently observed in northern Asia, Europe, North and South America, Australia, and New Zealand. It can be found in India's high temperate highlands above 2500 metres. The disease can have a negative impact on the export trade, particularly of seed tubers.

Symptoms: The disease has no foliar or above-ground symptoms. The first symptoms of powdery scab visible to the naked eye are small, usually purple-brown pimple-like swellings up to 2 mm in diameter seen most frequently at the rose (distal) end of young tubers. Powdery scab lesions, unlike normal scab, are spherical, elevated, packed with a powdery mass of spores, and surrounded by perforated epidermal layers (Fig. 87). These lesions have a scab-like appearance and are filled with fine brown powder consisting predominantly of cystosori. Cystosori, also known as sporeballs, are aggregates of individual cysts (resting spores) of the pathogen. Small galls may develop on the roots of affected plants. In storage, the infected tuber may shrivel and develop rot symptoms.

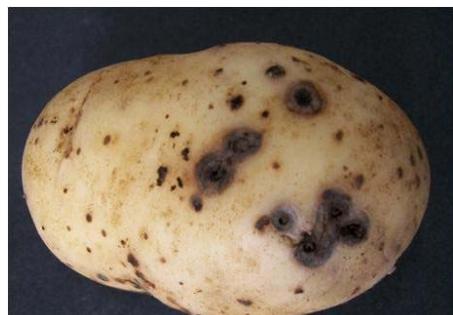


Fig. 87: Powdery scab lesions on tuber

Causal organism(s) & Key characteristics: Powdery scab is caused by *Spongospora subterranea*. *S. subterranea* survives, possibly for many years in a quiescent form as uninucleate or often binucleate thick-walled resting spores that are so resistant to adverse conditions that they can survive passage through the alimentary canal of farm animals. Individual resting spores are about 4 mm in diameter, but they are normally clumped together as cystosori. Each cystosorus is an irregular sphere or ovoid, 19–85 mm across, and usually has a hollow centre often with smaller pores between adjacent sori. These spaces contribute to the spongy nature of the spore

balls, from which the generic name, *Spongospora*, is derived. Potato mop-top furovirus is also transmitted by *S. subterranean*.

Disease cycle and favourable conditions: As sporangia grow in infected potato tubers, the pathogen survives the winter. It can live for up to 6 years in the soil. Under favourable conditions, and only in the presence of liquid water, the resting spores germinate, each one releasing usually, and possibly always, a single uninucleate zoospore through a circular hole in the wall of the sorus. The pathogen's zoospores penetrate the host's roots, stolons, and tubers, forming a multinucleate sporangial plasmodium. The plasmodium generates sporangia, which produce up to eight secondary zoospores. The zoospores re-infect the host tissue, and under perfect conditions, multiple generations of zoospores can be produced in a single season. The plasmodium produces resting spore in the tuber, which can survive the winter and persist in the tuber and soil for a long time. Other plants affected by the disease include oilseed rape, sugarbeet, spinach, and weeds such as chickweed, poppy, nettle, and fat hen. The time for tuber infection and gall formation is about three weeks at 16-20°C. The incidence is high in fields having pH range 4.7-7.6.

Management

- ❖ Use healthy seed from a disease-free environment, avoiding situations that lead to flooding of the fields by good drainage and following crop rotation with non-solanaceous hosts.
- ❖ Fungicides are ineffective in the field for controlling powdery scab.
- ❖ Do not use manure from animal fed infected tuber.
- ❖ Powdery scab can be reduced by planting trap crops like *Datura stramonium* right before planting potatoes.

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Name of disease: WHITE MOLD/ SCLEROTINIA STEM ROT

Geographical origin, distribution & crop losses: The earliest record of white mold disease incidence on potatoes corresponds to the year 1979. The first record of this disease in India is from Kota Rajasthan in the year 1989. During the year 2009 in some parts of West Bengal the disease has been reported to cause 60% loss to the standing crop. The disease can reduce yield by as much as 50% by causing the death of the above-ground portion of the plant. White mold is frequently found on beans, peas, carrots, and lettuce, in addition to potatoes.

Symptoms: Lesions commonly begin at the junctions of the stem and branches, or on branches and stems that come into touch with the soil. If moisture is available for several hours, these get swiftly covered by a white cottony growth that can spread quickly to surrounding stems and leaves. Lesions can girdle stems and cause foliage to wilt as they become larger. The girdling canker on the stem causes vines in the field to quickly wilt

and perish. The diseased area may develop a white mycelium covering (Fig. 88). After infection, the stem becomes light brown to white and becomes brittle. Sclerotia are typically found inside dried stalks. After row closure, little inverted trumpet-shaped structures known as apothecia can be found under the vine cover.

Causal organism(s) & Key characteristics: The disease is caused by a fungus, *Sclerotinia sclerotiorum*. The fungus produces white and fluffy mycelia on the culture media. It overwinters as hard sclerotia in the soil. Tiny globose spermatia also appear in the mycelia mat in the rhizosphere of infected plant. Sclerotia germinate in the spring and create apothecia (Funnel shaped to flat), which are little inverted mushroom-like structures that shoot spores into potato leaf. These apothecia vary in colour ranging from pale, tan, pink, white to brown. Ascii are hyaline, eight spored and cylindroclavate. Ascospores are oval hyaline with narrow base and inflated apex. The importance of spores produced in the potato field for infection is greater than that of spores produced outside the field.



Fig. 88: *Sclerotinia* stem rot symptoms

Disease cycle and favorable conditions: Soilborne sclerotia near the surface germinate, forming either an apothecium or, if enough moisture and organic matter are available, a mycelial mat. The mycelium penetrates stems at the soil line and forms a white, fluffy mat on the stem and adjacent soil. The fungus invades plant tissues rapidly, entering the inner stem tissues and pith, where sclerotia are formed. Apothecia forcefully eject numerous ascospores at maturity. Ascospores may spread a considerable distance from the source, settle on lateral branches or leaf surfaces, germinate, infect, and cause lesions. Spores can also directly infect the stem. Sclerotia can also germinate and infect the plant directly. Cool temperatures (16-22°C) and high relative humidity (95-100%) favor disease development. Sclerotia are killed within 3-6 weeks in flooded fields. Heavy rainfall or irrigation induces apothecia production from sclerotia.

Management:

- ❖ Management of this pathogen is very difficult because of its wide host range and persistent sclerotia. However, cultural practices, such as removal and burning of infected plant debris, eradication of weed hosts may suppress the disease incidence.
- ❖ Crop rotation with non-susceptible hosts for four or more years will help minimize sclerotinia rot in subsequent potato plantings.
- ❖ By increasing spacing, reducing vine growth, and irrigating less frequently, it may be possible to manage the outbreak.
- ❖ Foliar applications of protectant fungicides at row closure to coincide with ascospore release can limit the disease.

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Name of disease: SCLEROTIUM ROT

Geographical origin, distribution & crop losses: The disease is serious in warm humid regions. The fungus was named *Sclerotium rolfsii* by Saccardo in 1911. *S. rolfsii* has an extensive host range; at least 500 species in 100 families are susceptible. The most common hosts are the legumes, crucifers, and cucurbits. This pathogen has been reported on potato in several countries such as India, Israel, Italy, and the United States of America.

Symptoms: Plants with sclerotium stem rot first show a moist decay at or just below the soil surface (Fig. 89). Stem lesions can spread up and down the stem, infecting all plant sections. Wilting and yellowing of the leaves are symptoms of stem infection. Usually, the plants wilt and bear chlorotic leaves. An appressed, cottony fanlike mycelial growth emerges over the soil surface, and minute round, tan sclerotia form in the older mycelia at the stem base and soil surface. Initially, infected tissues are soft, depressed, and brownish. As the dead cortical stem tissues dry out, xylem remains as fibrous strands. Stolons are the most common way for tubers to become infected. Lenticels may also facilitate the mycelial entry that establishes infection in the tubers. Fresh lenticel lesions produce moist, firm and cheesy lesions. These lesions are easily dislodged and leave a cavity. The circular lesions become white and chalky after drying.



Fig. 89: Symptoms of *Sclerotium* rot on collar region

Causal organism(s) & Key characteristics: The causal fungus *Sclerotium rolfsii* bears white hyaline septate mycelium on the surface of incubated tubers and on PDA. Light to dark brown sclerotia 1 to 2 mm in diameter develop from the mycelium which are initially white but turn brown at later stages. Older mycelia usually form strands of pigmented hyphae. Sclerotia are numerous, round, 0.4-2.0 mm in diameter, white when young, then tan, and dark brown when old. The basidiospores of approximately 3.5-5 X 6-7 µm, elliptical to obovate, appear. Thick clamp connection is observed in the developing mycelia.

Disease cycle and favourable conditions: The fungus is soilborne as sclerotia or mycelia on dead and decaying organic matter. Sclerotia permit long-term survival but possess low energy reserve. They produce short-lived mycelia unless a suitable living or dead plant part is available. Mycelia infect seed tubers, sprouts, grown plants at any stage, and tubers. As an energy supply is exhausted, the mycelia aggregate, and sclerotia are formed. The fungus survives in the soil in off seasons. Hot temperatures (80° to 90°F) and moist soil surfaces enhance germination and sclerotia infection. Only hot climate growth locations are thought to be affected by Sclerotium stem rot. The fungus can infect both living and dead vines. If the fungus is present, extensive tuber losses might begin within a few days after harvest.

Management

- ❖ The fields infested with *S. rolfsii* should be avoided in succeeding seasons and should follow crop rotation with non-fleshy crops.

- An integrated package involving sowing of sorghum in *rabi* + deep ploughing in summer + FYM application @ 10 t/ha at planting + tuber treatment with carboxin (0.2%) followed by *T. harzianum* (1%) + soil application of *Neem* cake (2 q/ha) has been found effective in managing *sclerotium* wilt.

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Name of disease: **BACTERIAL WILT AND BROWN ROT**

Geographical origin, distribution & crop losses: The disease was first reported in south USA on tomato plants in the late nineteenth century and is believed to have spread to other countries through infected plant materials (e.g. potato tubers). Recently, Brazil is considered as one of the hypothetical centres of origin of *R. solanacearum* because of high genetic variability of *R. solanacearum* as brown rot ecotypes is autochthonous in South America, more precisely in Brazil and Peru. The disease is widespread in tropical and subtropical parts of Asia, Africa, South and Central America and in some soils and waterways in Europe and Australia. Bacterial wilt has been estimated to affect potato crop in 3.75 million acres in approximately 80 countries with global damage estimates exceeding \$950 million per year. Strains of this pathogen affect more than 450 plant species in over 54 botanical families throughout the world, including a wide range of crop plants, ornamentals and weeds. In India, losses up to 75 per cent have been recorded under extreme conditions.

Symptoms: The earliest symptom of the disease is slight wilting in leaves of top branches during hot sunny days. The leaves show drooping due to loss of turgidity followed by total unrecoverable wilt (**Fig. 90a**). In well-established infections, cross-sections of stems reveal brown discoloration of infected tissues (**Fig. 90b**). In advanced stages of wilt, cut end of base of the stem may show dull white ooze on squeezing. Bacterial wilt in field can be distinguished from other fungal wilts by placing the stem cut sections in clear water. Within a few minutes, a whitish thread like streaming can be observed coming out from cut end into water (**Fig. 90c**). This streaming represents the bacterial ooze exuding from the cut ends of colonized vascular bundles. The same test can also be carried out to see infection in tuber.

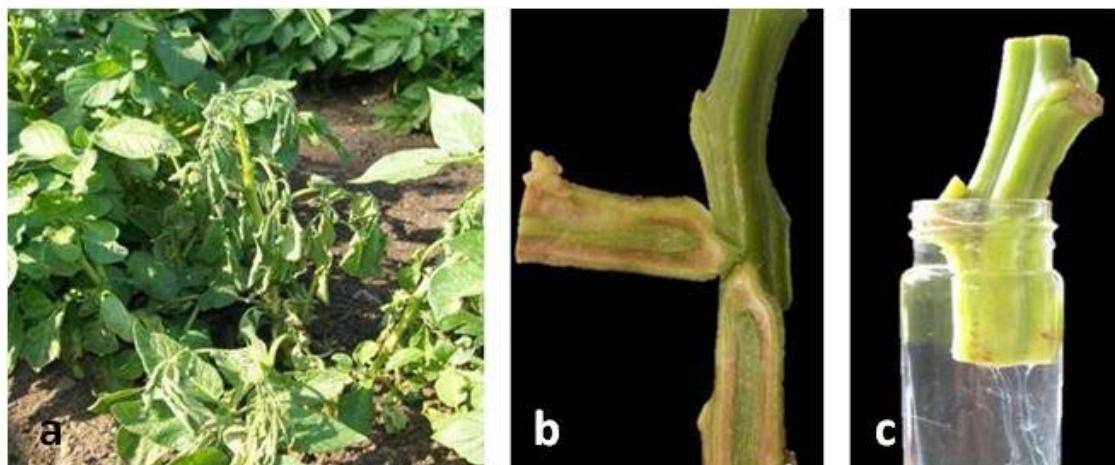


Fig. 90: Symptoms of bacterial wilt (a) wilting of plants infected with *R. solanacearum*, (b) brown discoloration of stem tissues, and (c) bacterial streaming in clear water from cut section of potato stem

Symptoms on potato tubers appear as vascular rot and pitted lesions formed on tuber surface (**Fig. 91a**). In vascular rot, the vascular tissues of a transversely cut tuber show dirty white glistening sticky drops of bacterial ooze appearing from the brownish vascular ring within about 2-3 minutes (**Fig. 91b**). Another type of symptom is observed as lesions formed on tuber. Such lesions are produced due to infection of the bacterium through lenticels (skin pore). Initially, water-soaked spots develop which enlarge in the form of pitted lesion. In advanced stages of wilt, bacterial mass may ooze out from eyes (**Fig. 91c**). Such sprouts may carry soil glued with the bacterial ooze. The tubers may not rot in storage and may not show vascular browning but still carry the pathogen. Such latently infected seed potato tubers may serve as a fresh source of inoculum.



Fig. 91: Symptoms of *R. solanacearum* infection on potato tubers (a) external symptoms on tubers, (b) vascular browning of tubers, and (c) oozing of bacterial mass through eye

Causal organism(s) & Key characteristics: *Ralstonia solanacearum* is a Gram-negative, rod-shaped, strictly aerobic bacterium that measures $0.5\text{-}0.7 \times 1.5\text{-}2.5 \mu\text{m}$ in size. This is non-spore forming, non-encapsulated, nitrate reducing and ammonia forming bacterium. The pathogen under oxygen stress conditions in culture media shift to avirulent form. Lipopolysaccharides of the pathogen play an important role in determination of virulence. Virulent isolates on tetrazolium chloride medium develop fluidal irregular shaped colonies with white to pinkish centre whereas avirulent types produce small round, dark red dry colonies.

Strains of *R. solanacearum* have conventionally been classified into five races (related to the ability to wilt members of the family *Solanaceae* (r1), banana (r2), potato and tomato in temperate conditions (r3), ginger (r4) and mulberry (r5), and six biovars (bvs) (metabolic profiles related to the ability to metabolize a panel of three sugar alcohols and three disaccharides). Based on variation of DNA sequences, *R. solanacearum* has been classified into four phylotypes in a species complex namely, Phylotype I (strains of Asiatic origin), Phylotype II (American origin), Phylotype III (African highlands and Indian ocean) and Phylotype IV (Strains in Indonesia). Recently, it has been reclassified into three distinct species: *R. solanacearum* (Phylotype II), *R. pseudosolanacearum* (Phylotype I and II) and *R. syzygii* (Phylotype IV). *Ralstonia syzygii* comprise three subspecies: subsp. *syzgii* found on clove, sub sp. *celebesensis* found on banana and sub sp. *indonesiensis* found on potato, tomato, capsicum and clove.

Disease cycle and favorable conditions: Infected tubers and plant debris in infested soil are two major sources of inoculum. The pathogen infects roots of healthy plants through wounds. Nematodes such as *Meloidogyne incognita* which affect potato roots and tubers increase wilt incidence. Inoculum potential of about 10^7 cfu/g soil favours infection which however is dependent on other predisposing factors. Race 1 has greater ability to survive in soil than race 3 because of the better competitiveness; wide host range and higher aggressiveness of race 1. Mean soil temperature below 15°C and above 35°C do not favour the disease development. Soil moisture influences the disease in at least four ways; (i) increasing survival of the bacterium in the soil, (ii)

increasing infection (iii) increasing disease development after infection, and (iv) increasing exit of the bacterium from host and spread through the soil. *Ralstonia solanacearum* is capable of causing brown rot in a wide range of soil types and levels of acidity. In majority of the cases, the disease has been reported in acidic soils (pH 4.3 to 6.8) and only in a few cases in alkaline soils. Several other factors that affect pathogen survival in soil and water also affect disease development. The soil type and physicochemical properties have significant influence on survival of the pathogen. Soils having high clay and silt content with higher water holding capacity are favourable for long survival while high sand contents disfavor its survival. Also, soil moisture and temperature exert a combined effect on survival of the pathogen. The congenial conditions for slow decline of population and virulence for race 1 and 3 are temperature between 10-30°C, soil moisture between 20-60 WHC, heavy soils and aerobic conditions.

Management

- ❖ The control of bacterial wilt has proven to be very difficult because of both the seed and soil borne nature of the pathogen and especially in the case of race 1 due to its broad host range. Chemical control is nearly impossible. Soil fumigants have shown either slight or no effects. Antibiotics such as streptomycin, ampicillin, tetracycline and penicillin hardly have any effect; in fact, streptomycin application increased the incidence of bacterial wilt in Egypt.
- ❖ Use of healthy planting material can take care of almost 80% of bacterial wilt problem. Tubers should not be cut since the cutting knife spreads the disease and cut tubers can contact disease from soil easily.
- ❖ Following 2-3-year crop rotation using crops like maize, cereals, garlic, onion, cabbage and sanai (sun hemp) can help in reduction of the disease. Care should be taken not to rotate vegetables like brinjal, ginger, chillies and other solanaceous crops which may act as alternate hosts. As the pathogen perpetuates in the root system of many weeds and crops, the fields should be cleaned of weeds and root/foliage remnants. Soil solarisation and deep ploughing of fields together during summer season in subtropical plains can help in reduction of field inoculum.
- ❖ Potato cultivars developed in Colombia with a *Solanum phureja* and *S. demissum* background showed resistance to *R. solanacearum*, but the race and strain diversity of the pathogen made it difficult to utilize these in other countries. The absolute control of bacterial wilt, at present, is difficult to achieve, however, economic losses can be brought down considerably using the eco-friendly package of practices.
- ❖ Soil application of stable bleaching powder @ 12 kg/ ha mixed with fertilizers at planting gives good control of bacterial wilt. Biological control of bacterial wilt has been investigated both in India and elsewhere. The use of antagonists such as *Pseudomonas fluorescens*, *Bacillus* spp., avirulent *P. solanacearum* and actinomycetes have been found to be effective against the pathogen under controlled conditions. *Bacillus subtilis* strain B5 has been reported to be effective against bacterial wilt pathogen.
- ❖ Overall an integrated approach involving use of pathogen free seed potato obtained from disease free areas, reduction of field inoculum through sanitation, soil solarization and crop rotation, growing crop under right environmental conditions, application of stable bleaching powder in soil can help in effective management of the bacterial wilt of potato.

Name of disease: SHOOT PROLIFERATION OF POTATO

Shoot proliferation is phytoplasma associate disease of potato recently detected in Odisha state of India in commercial potato varieties

Symptoms: Little leaf, Profuse shoot proliferation in variety Kufri Khyati and Kufri Surya (Fig. 92)

Distribution: Bhubaneshwar (Odisha)

Causal organism: 16SrVI-D subgroup phytoplasma (*Candidatus Phytoplasma trifolii*)

Disease cycle and Epidemiology: Different weeds and leafhoppers vector are identified as carrier and vector of the disease in India



Fig: 92. Symptoms of phytoplasma associated diseases in potato: a: little leaf, b: shoot proliferation; c: small and malformed tubers

Management: Early detection by molecular methods, leafhopper control and varietal resistance

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Name of disease: Common Scab

Geographical origin, distribution & crop losses: Common scab occurs in most potato producing areas in Africa, Asia, Europe, North and South America. In India, it was known to occur in Lahaul Valley (Himachal Pradesh) in severe form since 1969; its frequent occurrence in plains was reported in 1979-1980. Afterwards, it became a major problem in almost all agro climatic zones of India. Now, the disease has covered almost all the potato growing areas of the country and is posing a serious threat to successful potato cultivation. Common scab causes superficial lesions on surface of potato tubers and affects quality of the produce. The affected tubers fetch low price in market due to poor appearance and because deeper peeling is required before consumption. Seed lots exceeding 5 per cent incidence is rejected by seed certification agencies (in India) causing huge loss to seed industry.

Symptoms: Scab begins as small reddish or brownish spot on the surface of the potato tubers and its initial infection takes place during juvenile period of tubers. Infection takes place mainly through lenticels and surrounding periderm turns brown and rough. Lesion becomes corky due to elongation and division of invaded cells. Under Indian conditions multiple kinds of symptoms have been recorded and they are grouped as (1) a mere brownish roughening or abrasion of tuber skin (2) proliferated lenticels with hard corky deposition, might lead to star shaped lesion (3) raised rough and corky pustules (4) 3-4 mm deep pits surrounded by hard corky tissue (5) concentric series of wrinkled layers of cork around central black core (**Fig. 93**).

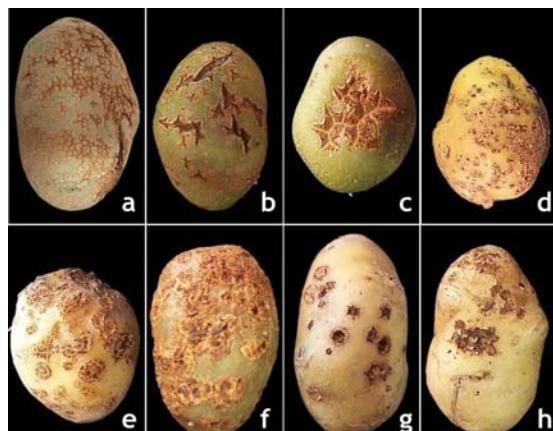


Fig. 93: Different types of scab symptoms caused by *Streptomyces* species on potato tubers

Causal organism(s) & Key characteristics: At least 13 different *Streptomyces* spp. have been found to cause common scab on potato worldwide. The prominent among them are *Streptomyces scabies* (Thaxter) Lambert and Loria, *S. acidiscabies* Bambert and Loria, *S. turgidiscabis* Takeuchi, *S. collinus* Lindenbein; *S. griseus* (Krainsky) Waksman & Henria Henria, *S. longisporoflavus*, *S. cinereus*, *S. violenceoruber*, *S. alborgriseolus*, *S. griseoflavus*, *S. catenulae* and others. *Streptomyces* are bacteria which resemble fungi due to formation of vegetative substrate mycelium that develop aerial filaments. However, the filaments are of smaller dimensions than the true fungi. These filaments produce spores through fragmentation. *Streptomyces* spp. may be pathogenic or non-pathogenic. The pathogenic species produce thaxtomins which are phytotoxins and cause hypertrophy and cell death. Considerable variation exists within the pathogen with respect to their pigment production in media, colour and shape of sporulating filaments and use of specific sugars. The identification and taxonomy of *Streptomyces* spp. has been based on morphological and physiological characteristics combined with thaxtomin production and pathogenicity tests *in-vitro* and *in-vivo*.

Disease cycle and favorable conditions: Potato is physiologically most susceptible to *Streptomyces* sp. in the period following tuber initiation. *Streptomyces* sp. infects the newly formed tubers through stomata and immature lenticels. Once the periderm has differentiated, tubers are no longer susceptible to the pathogen. The pathogen is both seed and soil borne. It can survive in soil for several years in plant debris and infested soil. Soil conditions greatly influence the pathogen survival. Favorable conditions include pH between 5.2 to 8.0 or more, temperature in the range of 20 to 30°C and low soil moisture. The pathogen is aerobic in

nature and maintaining high soil moisture for 10 to 20 days after tuber initiation can help in reducing the common scab. However, scab outbreaks have been reported in irrigated or wet soil conditions in northern Europe, Israel, and Canada. The organism is a tuber-borne and is well-adapted saprophyte that persists in soil on decaying organic matter and manure for several years. Infected tubers serve as source of inoculum in the field, giving rise to infected progeny tubers. The pathogenic *Streptomyces* species are both soil and tuber-borne. Tuber-borne inoculum is likely to be involved in the distribution of new strains or species.

Management

The pathogen is difficult to eradicate because of long survival both on seed tubers and in soils. In general, common scab can be managed by use of disease free seed tubers, tuber treatment with boric acid (3% for 30 min.) before cold storage, keeping the moisture near to field capacity right from tuber initiation until the tubers measure 1 cm in diameter, following 3-4 year crop rotation with wheat, pea, oats, barley, soybean, sorghum, bajra, green manuring and deep ploughing the potato fields in April and leaving the soil exposed to high temperatures during summer (May to June) in the North Indian plains.

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Name of disease: BLACK LEG AND SOFT ROT

Geographical origin, distribution & crop losses: Bacterial soft rot of potato is found wherever potatoes are grown. The disease affects the crop at all stages of growth, but it is more serious on potato tubers under poor storage conditions especially in warm and wet climate. Bacterial soft rot can cause significant loss of potato tubers at harvest, transit and storage. Losses due to poor handling of the produce, poorly ventilated storage or transit may go up to 100 per cent. Soft rot bacteria usually infect potato tubers which have been damaged by mechanical injury or in the presence of other tuber borne pathogens. Bacterial soft rot develops much faster under warm and humid conditions. The disease also results in blackleg of foliage during the crop growing season.

Symptoms: Initially a small area of tuber tissue around lenticels or stolon attachment point becomes water soaked and develops soft lesions (**Fig. 94a**). Under low humidity, the initial soft rot lesions may become dry and sunken. Under high humidity, the lesions may enlarge and spread to larger area. Tubers in advanced stages of decay are usually invaded by other organisms and the decaying tissue becomes slimy with foul

smell and brown liquid ooze. The tuber skin remains intact and sometimes the rotten tubers are swollen due to gas formation. At harvest, many small rotten tubers with intact skin can be seen. The infected seed tubers rot before emergence resulting in poor stand of the crop. In cooler regions, another kind of symptoms called black leg phase develops from soft rot infected seed tubers. The affected haulms become black at collar region just above the ground. Infected plants develop yellowing, start wilting and die early without producing any tubers. Water soaked lesions develop on succulent stems, petioles, and leaves. On stem and petioles, the lesions first enlarge into stripes, turn black (Fig. 94b) and then invade the affected parts causing soft rot and toppling of the stem and leaves.

Causal organism(s) & Key characteristics: *Pectobacterium atrosepticum* (van Hall) (syn. *Erwinia carotovora* sub sp. *atroseptica*), *Pectobacterium carotovorum* sub sp. *carotovorum* (Jones) (syn. *Erwinia carotovora* subsp. *carotovora*), *Pectobacterium carotovorum* subsp. *brasiliense* (*Erwinia carotovora* sub sp. *brasiliense*) (Pcb), *Pectobacterium wasabiae* (*Erwinia carotovora* sub sp. *wasabiae*) (Pwa) and several *Dickeya* spp. (*Erwinia chrysanthemi*), including *D. dianthicola* (*Erwinia chrysanthemi* pv. *dianthicola*), *Dickeya dadantii*, *Dickeya zeae* (*Erwinia chrysanthemi* pv. *zeae*) and the new species *Dickeya solani* are known to cause potato blackleg disease in field and tuber soft rots in storage and in transit. *P. atrosepticum*, the primary enterobacteria causing soft rots, produce pectolytic enzymes and degrade pectin in middle lamella of host cells, breakdown tissues and cause soft rot and the decay. The decaying tissue become slimy and foul smelling and brown liquid oozes out from the soft rot affected tubers. About 1500 strains of pectinolytic *Erwinia* have been isolated from infected plants and tubers. The pathogen produces certain volatile compounds such as ammonia, trimethylamine and several volatile sulfides and early detection of such volatile compounds in storage could be used as a method to detect the disease at initial stage.

Disease cycle and favorable conditions: Soft rot bacteria may be carried latently deep inside the tuber in lenticels, wounds and on surface of tubers without any visible symptoms and spread to healthy tubers in stores, during seed cutting, handling and planting. Water film on surface of tuber which cause proliferation of lenticels and creates anaerobic conditions and injury on surface of tuber predispose potatoes to soft rot. From soft rot infected seed tubers bacteria may enter vascular tissues of developing stems and can develop black leg under favorable conditions. From black leg infected plants, the pathogen can reach daughter tubers through stolons and initiate tuber decay at the site of tuber attachment. Decaying tubers in soil could serve as source of contamination for healthy tubers. The threshold level for disease development is about 10^3 cells of *E. carotovora* sub sp. *atroseptica* per tuber. Tubers harvested in wet soil, poor ventilation in transit and storage promotes the rot.

In warm climates, where one potato crop follows another or where only short rotation cycles are applied, the bacteria can pass easily from one crop to the next, especially in poorly drained soil. The bacteria can be disseminated in the potato fields by irrigation water, insects, rain or bacterial aerosols. The pathogen may also spread through water during washing of the produce with contaminated water. Soft rot causing bacteria spread easily from diseased to healthy tubers during storage, handling and grading. Insects especially maggots of *Hylemyia* species may also transmit the bacteria from one tuber to another.

There are controversial reports on survival of *Erwinia*s in soil. In temperate zones, the bacteria can survive the winter on plant residues, however, no survival has been observed in fields rotated with non-hosts



Fig. 94: a) Soft rot symptoms on tubers b) black leg symptoms on stem

crops. The bacteria can survive at places where rotten potatoes and vegetables are dumped.

Management

An integrated approach involving practices like planting of whole seed potato or well suberized seed pieces in well-drained soil with temperature around 10 to 13°C at less planting depth, tuber treatment with 3.0 per cent boric acid or 0.05 percent copper sulphate, restricting nitrogen dose to minimum (150 kg/ha), application of stable bleaching powder before planting and during last irrigation, crop rotation following green manure-potato – wheat, avoiding bruises and cuts to potato tubers during harvest, handling, and proper aeration during storage and transit can minimize soft rots. Adjusting planting time to avoid hot weather during plant emergence and harvesting the crop before soil temperature rises above 28°C are also recommended to minimize the losses due to soft rot.

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Name of disease: POTATO VIRUS X (PVX)

Geographical origin, distribution & Crop losses: PVX is found wherever potatoes are grown. In nature it is largely confined to members of the family *Solanaceae*. Yield losses of 15–20% can occur with widespread infection of PVX. The virus interacts with PVY and PVA to cause more severe symptoms and still higher yield loss than either virus alone.

Symptoms: Plants infected with PVX often remain symptomless. When symptoms are expressed, there is a pattern of light and dark green on leaflets, the lighter, small, irregular blotches occur between the veins. Only occasionally does leaf distortion, rugosity, necrotic spotting or stunting occur. PVX can interact with PVY and PVA to cause more severe symptoms such as rugose mosaic (PVX+PVY) and crinkle mosaic (PVX+PVA) resulting in higher yield loss.

Causal organisms & Key characteristics: PVX has filamentous virions of 515x13 nm and single stranded RNA. PVX belongs to the family *Alphaflexiviridae* and the order *Tymovirales*. It is the type species of the genus *Potexvirus*. Strains of PVX have been classified into four groups according to their reactions with the dominant resistance genes Nb and Nx and with the extreme resistance gene Rx. Group 1 strains cause a hypersensitive response in the presence of Nb or Nx, group 2 only with Nb, group 3 only with Nx and group 4 with neither and fails to infect plants carrying Rx gene. Based on serology, two serotypes can be distinguished: serotype PVX^O (common or worldwide distributed) and PVX^A (restricted to some regions in Peru and Bolivia).

Disease cycle and favorable conditions: Transmission of PVX is by contact, either plant to plant or by humans, animals or machinery passing through a crop. PVX is highly contagious. Once attached to a surface such as clothing the virus can remain infective for many hours provided the surface remains wet. The virus accumulates in tubers, and the process of cutting seed tubers can spread the virus from one tuber to

another. PVX is not transmitted by true seed or by aphids. PVX can spreads rapidly in poorly cared crop resulting in up to 50% or more infection with in 3-4 cycles of cultivation.

Name of disease: POTATO VIRUS Y (PVY)

Geographical origin, distribution & Crop losses: PVY is one of the most economically important viruses of potato worldwide. It can cause up to 80 % yield reduction. PVY^O occurrence is worldwide. However, PVY^N and PVY^{NTN} strains are limited to certain countries, and therefore, are of quarantine significance. PVY^Z and PVY^E have also been reported but these are considered to be of lesser significance in potato production. In addition to these strains, recombination led to the evolution of new strains of PVY like PVY^{NTN}, PVY^{NW} and PVY^{NTN-NW}. PVY^C strain has been reported from India, Australia and Europe.

Symptoms: Generally, the symptoms vary with the strains of the virus and variety of potato. In general, the affected plants are stunted with mild or severe mosaic and veinal necrosis. Original strain of PVY^O causes mosaic, necrotic spots, mild rugosity and drying of leaves whereas, PVY^C usually causes stipple-streak or necrotic veins/stars. PVY^{NTN} strains cause severe superficial tuber necrosis and may also causes necrotic foliar symptoms (**Fig. 95**).

Causal organisms & Key characteristics: PVY are flexuous, long filamentous, 740 x 11 nm sized particles. PVY is a type member of the genus *Potyvirus* of the family *Potyviridae*. The potato PVY isolates are classified into different strain groups based on their mosaic or necrotic symptoms developed on tobacco and resistance gene interaction involving elicitation of specific HR genes. Initially, they were divided into three strains: PVY^C, PVY^O, and PVY^N but later two more strain groups PVY^Z and PVY^E were added. PVY^O and PVY^N are the most frequent in potato whereas PVY^C is not common in potato fields and thus of comparatively less economic importance. PVY^Z and PVY^E have also been reported less frequently.

Disease cycle and favorable conditions: Infected seed tubers or seed material is the main sources of PVY inoculum in tropical countries. Aphids (*Myzus persicae*) probing on potato plants are potential vectors of PVY which transmit the virus to other healthy plants. Tubers formed by the infected plant act as primary source of inoculum for the next year crop. Further, a combination of high humidity (80-90%) and moderate temperature (25-30°C) increase virus transmission by aphids. However, the transmission of this virus is reduced significantly when RH falls below 50%.

Name of disease: POTATO VIRUS A (PVA)

Geographical origin, distribution & Crop losses: PVA has worldwide distribution. In India it is most often observed as combined infections with PVX and PVY. PVA alone can cause 30-40 % yield reduction while it is still higher in case of co-infections with PVX and PVY.

Symptoms: Potato virus A (PVA) causes mild mosaic symptoms somewhat similar to those caused by Potato virus X (PVX). However, unlike PVX the mottles caused by PVA appear on the veins and the infected leaves look shiny. Infected plants show an open habit. Severe leaf crinkle may be produced in combination with PVX.

Causal organisms & Key characteristics: PVA virus particles are flexuous 730 x 11 nm long filaments. It is serologically closely related to PVY. PVA belongs to genus *Potyvirus*, family *Potyviridae* with single stranded positive sense RNA. Three strain groups of Potato virus have been reported based on their response in potato. PVA^U caused systemic necrosis in potato cv. King Edward, which carries the gene *Na_{KE}* for hypersensitivity to PVA, whereas PVA^M caused mottle symptoms without necrosis. In other potato cultivars carrying *Na* gene,



Fig. 95: Necrotic lesions due PVY infection

PUA^{U} and PVA^{M} caused systemic necrosis. PVA^{B11} caused no hypersensitive response in potato cultivars carrying the gene Na or Na_{KE} . Thus, PVA^{U} , PVA^{M} and PVA^{B11} represented distinct strains of PVA, namely $\text{PVA}^{\text{-1}}$, $\text{PVA}^{\text{-2}}$ and $\text{PVA}^{\text{-3}}$, respectively.

Disease cycle and favorable conditions: At least seven aphid species are capable of transmitting PVA including *Aphis frangulae*, *Macrosiphum euphorbiae*, and *Myzus persicae*. The virus is non-persistent and is lost from aphids as they go through their life cycle. As with PVY, the virus can be acquired rapidly from an infected plant (<1 minute) and transmitted equally rapidly. The transmission and life cycle of infected tubers are almost similar to PVY. PVA naturally infects only potato crop however, this virus can infect a few *Solanaceae*ous species.

Name of disease: POTATO VIRUS S (PVS)

Geographical origin, distribution & Crop losses: PVS occurs worldwide. It is the most frequently found virus in potato. An incidence of 8.5 to 99.5% has been recorded in Indian potatoes at different locations. Reduction in tuber yield is very low but may go up to 10-20% or more when combined with PVX.

Symptoms: PVS^{O} is symptomless on most potato cultivars, with occasional mild leaf symptoms of rugosity, vein deepening and bronzing. PVS^{A} has been found to produce stronger symptoms, including premature senescence and loss of leaves, especially in secondarily infected plants. However, symptoms such as rugosity, mosaic, stunting, bronzing, vein deepening, early drop of leaves, and fine necrotic spots on lower leaves have also been reported occasionally in some cultivars.

Causal organisms & Key characteristics: PVS belongs to the genus *Carlavirus*, family *Betaflexiviridae*. PVS consists of flexuous particles containing a single-stranded, positive-sense RNA genome of approximately 8.4Kb. The genomes of most *Carlavirus* contain positive single-stranded RNA of 5.8 to 9kb and two subgenomic mRNAs of 2.1-3.3 kb and 1.3-1.6 kb. Two strain groups of PVS have been recognized viz., PVS^{O} (Ordinary) and PVS^{A} (Andean) based on non-systemic and systemic infection in *Chenopodium* spp. Both PVS^{O} and PVS^{A} produce chlorotic local lesions on sap inoculation to *C. amaranticolor* and *C. quinoa* and in addition PVS^{A} induce systemic chlorosis. These two strains can also be identified by the differences in nucleotide sequences or amino acid sequences of the 7K protein, CP, and nucleotide-binding (11K) protein at the genetic level.

Disease cycle and favorable conditions: In the field, PVS is transmitted primarily by contact than by aphids. PVS can be transmitted by *Aphis fabae*, *A. nasturtii*, *Myzus persicae*, and *Rhopalosiphum padi* in a non-persistent mode or via vegetative propagation or naturally mechanical inoculation to the *Solanaceae* and *Chenopodiaceae*. PVS usually spreads slowly in the field. Spreading of PVS^{A} might faster than PVS^{O} , because of higher virus concentrations and additional transmission by *Myzus persicae* and other aphid species. Tubers also carry the virus. PVS being occurs as most frequent natural contaminant along with PVX, and/or PVY and PVM. The virus is transmitted through contact and by several aphid species in a non-persistent manner.

Name of disease: POTATO VIRUS M (PVM)

Geographical origin, distribution & Crop losses: Potato virus M (PVM) has worldwide distribution. It can cause 15-45% reduction in potato yield in heavily infected varieties.

Symptoms: PVM causes mottle, mosaic, crinkling, rolling of leaves and stunting of shoots. Severity of symptoms greatly depends on the combination of potato cultivars and PVM isolates.

Causal organisms & Key characteristics: PVM has a single-stranded, polyadenylated, positive-sense genomic RNA of appropriately 8.5 kb in length. PVM is a member of the genus *Carlavirus* of the family *Flexiviridae*. As per our literature search there are no reports on strains of PVM.

Disease cycle and favorable conditions: The main sources of PVM inoculum are infected seed tubers or seed material. It is experimentally sap transmissible. Aphids are the main vectors which transmits PVM in non-persistent manner from infected to healthy potato plants. The produce from infected plants carry virus in tubers which act as source of inoculum for the next year and the cycle repeats. PVM has a very narrow host range and mainly restricts to potato. However, experimentally it can infect a few species of *Chenopodiaceae* and *Solanaceae*.

Name of disease: POTATO LEAFROLL VIRUS (PLRV)

Geographical origin, distribution & Crop losses: Potato leafroll virus (PLRV) is found wherever potatoes are grown. It is mostly found in the Solanaceae family. The infected plants may 50% or more reduction in yield. PLRV is probably the most important potato virus worldwide. According to one assessment, the virus is to blame for Global yield loss of 20 million tonnes.

Symptoms: The primary symptoms are confined to top young leaves, which usually stand upright, roll and turn slightly pale in certain cultivars (Fig. 96). Most varieties, however, develop reddish/pink colour in top leaves starting at the margins, sometimes accompanied with slight rolling of the leaflets. Secondary symptoms develop when the plants are grown from infected seed tubers. Such symptoms are rather prominent in older leaves, i.e. absent or less pronounced on younger top leaves. Infected plants have characteristic pale, dwarfed, and upright appearance with rolling of lower leaves that turn yellow, brittle and are leathery in texture. In some cultivars, a reddish or purple discolouration develops on the margins and underside of the leaves.



Fig. 96: Primary symptoms of PLRV on young leaves

Causal organisms & Key characteristics: PLRV is the second most important virus of potato globally and pertain to the family *Luteoviridae* and genus *Poletovirus*. PLRV has small, isometric virions (23-25 nm dia) which are primarily confined to the phloem of the infected plants. The genome of PLRV is monopartite, single stranded, non-enveloped with a positive sense RNA of 5882 nucleotides in length.

Disease cycle and favorable conditions: The virus is tuber borne, transmitted efficiently by aphid in a persistent manner. Primary infections are normally caused by the viruliferous aphids coming either from distant or nearby fields and/or diseased volunteer plants arising from the infected tubers. Potato plants infected with PLRV will produce infected tubers. If infected tubers are planted, they will give rise to infected plants. The green peach aphid (*Myzus persicae*) is the most efficient and important vector of PLRV. The virus is also spread by *Aphis gossypii*, *Macrosiphum euphorbiae* which transmits potato strains less efficiently. Virus accumulates in tubers and such tubers transmit the virus to the daughter tubers and to the foliage.

Name of disease: TOMATO LEAF CURL NEW DELHI VIRUS (TOLCNDV)

Geographical origin, distribution & Crop losses: Apical leaf curl disease of potato was observed in 1999 from Northern India. Association of a begomovirus was confirmed and it was found as a strain of *Tomato leaf curl New Delhi virus*. About 40-100 % of infection has been recorded in Indo-Gangetic plains and Hisar area of India with heavy yield losses in susceptible varieties. Significant decrease in size and number of tubers lead to marketable yield loss as high as 50% in early planted susceptible cultivars.

Symptoms: The affected plants show curling / crinkling of apical leaves with a conspicuous mosaic symptom. In case of seed borne infection, the entire plant shows the symptoms leaf distortion with mosaic accompanied with severe stunting of the plant (Fig. 97). The symptoms are so severe that it can be identified easily, but such symptoms are expressed only when the concentration of virus is very high. However, in many cases the symptoms are recovered as the maximum temperature fall below 28°C. If the virus concentration is less the leaves show rolling which is just like symptoms caused by Potato virus M (PVM).

Causal organisms & Key characteristics: Tomato leaf curl New Delhi virus-potato (ToLCNDV-potato) belongs to the genus *Begomovirus* within the family *Geminiviridae*. This virus is a variant of a bipartite begomovirus, *Tomato leaf curl New Delhivirus* (ToLCNDV) with altered properties is sap-transmissible and is able to infect potato. The virus has 93–95% sequence identity with ToLCNDV isolates and <75% sequence identity with other *Tomato leaf curl virus* isolates and *Potato yellow mosaic virus*.

Disease cycle and favorable conditions: Begomoviruses are transmitted by *Bemisia tabaci* in a circulative and persistent manner. Increase in populations of *B. tabaci* is a general characteristic of begomovirus disease epidemics which facilitates the spread of begomoviruses into and within and their transmission to host crops and from weed hosts. Increased populations of *B. tabaci* are associated conducive climatic conditions. Epidemiological models suggest a high density of vectors can favour the evolution of higher virulence in virus populations. The disease incidence is less in late planted crop in all the cultivars due to lower population of whitefly. The virus is also transmitted through seed tubers.

Name of disease: GROUNDNUT BUD NECROSIS VIRUS (GBNV)

Geographical origin, distribution & Crop losses: GBNV, a *Tospovirus*, causes stem necrosis disease in potato (PSND). Distribution of the tospovirus is worldwide. It is known to naturally infect potatoes from Argentina, Australia, Brazil and central India. Stem necrosis incidence was recorded up to 90% in some parts of Madhya Pradesh and Rajasthan and up to 50 % in Pantnagar. PSND is important in areas where thrips, the vector of GBNV, and the virus sources occur together. It is rather common in early crop of potatoes in Central/ Western plains and plateau regions of India. Losses up to 29 per cent have been reported by PSND.

Symptoms: Infected plants show extensive necrosis with formation of concentric rings or spots on leaves and stems (Fig. 98). Shoots that are not killed have stunted/rosetted appearance and chlorotic necrotic ring spots on leaves. Tubers on such plants may be few, sometimes small and rarely deformed yet without the virus. Primary infection shows as necrotic spots or rings on youngest leaflets. The spots may have concentric zonation and may be confused with early blight. Brown necrotic lesions develop on petioles and stems and within the stem. Death of one or more stem tops may occur or, in extreme cases, the whole plant may die. In secondary infection, plants arising from infected tubers are stunted and exhibit bunching of leaves. The leaflets are coarse and may desiccate and turn brown. Infected plants may appear normal or may show distortion or cracking but are usually small in size.

Causal organisms & Key characteristics: The virus has large roughly spherical, enveloped particles, ranging from 70-110 nm in diameter, covered with knob-like surface projections. GBNV belongs to the genus *Tospovirus* in the family, *Bunyaviridae*.



Fig. 97: Typical symptoms of ToLCNDV



Fig. 98: Characteristic symptoms of GBNV on potato leaves and stem

Disease cycle and favorable conditions: Several thrips species belonging to the genera *Thrips* and *Frankliniella* act as the virus vectors in a persistent manner but they can acquire the virus only as nymphs, with 4-9 days of latency and about 1 hr inoculation access. Viruliferous thrips move to early crop of potatoes from other preceding crops and infect the potato crop. The tospovirus has a wide host range including crops like tomato (*Lycopersicon esculentum*), tobacco (*Nicotiana* spp.), peanut (*Arachis hypogaea*), soyabean (*Glycine max*) and cotton (*Gossypium* spp.).

Name of disease: POTATO AUCUBA MOSAIC VIRUS (PAMV)

Geographical origin, distribution & Crop losses: Potato aucuba mosaic virus was first described and named by Quanjer in 1921. Probably distributed worldwide, but not common. The virus has been shown to occur in Europe (Ireland, Great Britain, Scandinavia, France, USSR, Hungary) as well as North America (United States, Canada), South Africa, Australia, New Zealand, Japan, and even in wild Solanum species from Peru. Khurana (2004) has reported 20-40% yield loss due to the incidence of PAMV on potato in plains of India.

Symptoms: It causes bright yellow spots on the lower leaves, later coalescing to form large yellow necrotic spots, often leading to systemic or top necrosis. The symptoms always appear on the lower leaves of the diseased plants and are confined there, but in the potato variety Irish Chieftain, a pronounced brilliant yellow mottle develops all over the plants. Tuber necrosis may also occur which starts from the heel end of the tuber and is favored by darkness arid high temperature. However, tubers from plants which develops mild foliar symptoms after inoculation with different isolates of the virus, showed internal necrosis in some varieties.

Causal organisms & Key characteristics: PAMV is a single stranded RNA (ssRNA) virus with filamentous particles about 580 nm long and 11 nm wide and belongs to genus Potexvirus; family *Alphaflexiviridae*; order *Tymovirales*. Virions not enveloped, filamentous ca. 580 x 11-12 nm.

Disease cycle and favorable conditions: It is transmitted by aphids (*Myzus persicae*) in the presence of 'helper' viruses of the PVY group in a non-persistent manner and readily transmitted by mechanical inoculation.

Name of disease: POTATO SPINDLE TUBER VIROID (PSTVD)

Geographical origin, distribution & Crop losses: Potato spindle tuber disease is widespread in North America, parts of Eastern Europe, Russia and China. Detection and elimination of the virus through systematic indexing over the last 10–20 years has significantly reduced PSTVd from commercial seed and ware production in North

America and parts of Eastern Europe. Yield losses vary with the isolate, cultivar and growing conditions. It could range from slight to serious (up to 64%). The virus can be transmitted through True potato seed (TPS).

Symptoms: In potato symptoms of PSTVd are strain/cultivar/environment-dependent and may vary from severe symptoms, which include reduction in plant size. Changes in plant growth is characterised by uprightness and clockwise phyllotaxy of the foliage, dark green and rugose leaves with mild to symptomless infection. Tubers may be reduced in size and may be misshapen to spindle and dumbbell shape with conspicuous prominent eyes which are evenly distributed.

Causal organisms & Key characteristics: Potato spindle tuber viroid (PSTVd) is a type species of Pospiviroidae. Viroids are the smallest-known infectious agents causing diseases in higher plants. They consist of small (241–399 nucleotides), single-stranded circular RNAs. Potato spindle tuber was the first member identified in 1971 as ‘viroid’.

Disease cycle and favorable conditions: It is highly contagious and stable and spreads through pollen, botanical seed and by contact which is mainly by use of machinery in the infected field. Experimental transmission by aphids, *Myzus persicae* from plants co-infected by PLRV has been reported.

Management of viruses

Diseases caused by viruses can be managed by adopting an integrated management as follows:

- 1) To avoid the spread of contagious viruses/viroid, strict sanitation in the field and in stores, right from harvest to planting.
- 2) Disinfect all field equipments by dipping in or washing them either with 3% trisodium phosphate or calcium hypochloride (1%) solution.
- 3) Essentially plant disease-free seed stocks from approved or reliable sources.
- 4) Rogue out the diseased plants carefully along with their tubers and dispose them away from the field at regular intervals during the growing season.
- 5) Dehaulming the crop before the aphids cross critical level to enforce rigid control of the insect vector and
- 6) Treat seed with imidacloprid (200SL) @ 0.04% (4ml/10lit) for 10 minutes before planting, spray imidacloprid @3ml/10 litre at emergence, provide second spray after 30 days of crop emergence, spray an alternative insecticide such as thiamethoxam (25WG) @0.05% at 50 days.

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Name of disease: POTATO CYST NEMATODES

Geographical origin, distribution & crop losses: The Andean Mountain of South America, the original home for potato is also the place of origin for PCN and introduced into Europe in the 1850's along with the late blight resistance breeding material. Later, it spread throughout the world through the introduction of late blight resistant varieties from Europe. As of now, PCN has turned into a major pest in the largest part of potato growing areas affecting >80 countries in the temperate, cooler parts of the tropical and subtropical regions of the world and cause more than 70% yield losses. In India, Dr. F.G.W. Jones first detected the PCN during 1961 from Vijayanagaram farm in Udhagamandalam, The Nilgiris district of Tamil Nadu and Tamil Nadu Government imposed domestic quarantine during 1971. Recently, the occurrence of this pest has also been reported from some parts of Himachal Pradesh, Jammu & Kashmir and Uttarakhand. Accordingly, the Government of India restricted the movement of potato seed tubers from the infested areas since 2018.

Symptoms: The disease caused by this nematode is often referred to as 'potato sickness'. Slightly infested crop does not show any above ground symptoms. However, heavy infestation shows localized poor growth of plants in small patches (**Fig. 99**) and wilting during hot parts of the day. Severely infested plants remain stunted with dull and unhealthy, as the season advances, the lower leaves turn yellow, brown and wither, leaving only the young leaves at the top and ultimately causes the premature death of the plant. The root system is poorly developed; the yield and size of the tubers are reduced considerably. Severely infested plants give little or no harvest and we can see the presence of white or yellow female nematodes sticking to the roots.

Causal organism(s) & Key characteristics:

- a) **G. rostochiensis:** Second-stage juvenile (J2) has short body length, stylet knobs short with prominent round, tail tapering to small with rounded terminus. The immature female is yellow/golden coloured and matured cyst is brown in colour. Cyst ovate to spherical in shape with protruding neck (**Fig. 100**).
- b) **G. pallida:** J2 has long body length, stylet knobs longer, basal knobs with distinct anterior projection, tail tapering uniformly and pointed. The immature female is white in colour and matured cyst is brown in colour. Cysts are ovate to spherical in shape with protruding neck (**Fig. 101**).



Fig. 99: Symptoms of field infected with potato cyst nematode

Disease cycle and favorable conditions: Juveniles (J2s) hatched out from the egg enters the root system of its host and begin feeding on the young potato roots and forms feeding site called syncytium. The J3 stage has well developed reproductive organs. At fifth stage (J5), the female ruptures the root cortex. Males

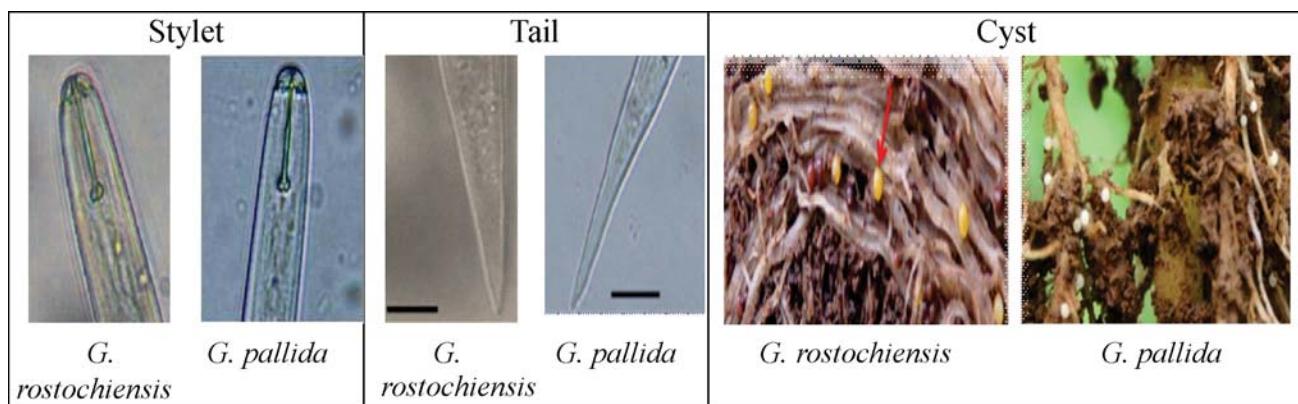


Fig. 100: Key characters of *G. rostochiensis* and *G. pallida*

develop at a similar rate in the same root as the females. After mating, the embryo develops within the egg as the formation of the J2 while still in the body. After completing the life cycle female dies, the body wall thickens to form a hard brown cyst that is resistant to adverse weather conditions. Mostly PCN complete the life cycle within 35-49 days. In general, one generation is completed in one crop season, *G. rostochiensis* use to complete two generation in one crop season due to shorter dormancy (45-60 days) while *G. pallida* has a dormancy period ranging from 60-75 days. For the development on the host, *G. pallida* require 10 to 18 °C, while *G. rostochiensis* requires 15 to 25 °C. Each cyst contains 200-500 eggs and is easily dislodged in soil at harvest. The eggs inside the cysts can survive in soil for up to 30 years even in the absence of a suitable host.

Management

- ❖ Restrict the movement of seed material from the PCN infested areas as well as use of diseased free certified seed to avoid the further spread of PCN to the new areas.
- ❖ Grow *Solanum andigena*, *S. sparsipilum*, *S. gourlayi*, *S. spegazzini* and *S. vernei* derived PCN-tolerant/ resistant varieties, such as Cara, Maris Piper, Kufri Swarna, Kufri Neelima, Kufri Sahyadri and Kufri Karan.
- ❖ Follow three to four years crop rotation with non-solanaceous crops involving potato, French beans, peas, radish, cabbage, cauliflower, turnip, garlic, carrot, green manure crop like lupin etc., intercropping of potato with French Beans (3:1), mustard (1:1), radish (2:1), biofumigation with radish leaves and polyethylene sheeting, incorporate *Brassica juncea* (Indian/brown mustard), and use of biological control agents such as *Pseudomonas fluorescens*, *Purpureocillium lilacinum*, *Trichoderma viride* reduce the PCN population. Grow susceptible potato cultivar as trap crop or wild trap plant *S. sisymbifolium* (non-tuber plant) to reduce PCN population. But the susceptible trap crops should be destroyed before completion of life cycle (35-40 DAP).
- ❖ Seed treatment with sodium hypochlorite (2%) solution can be used for decontamination of freshly harvested seed tubers.

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Name of disease: ROOT-KNOT NEMATODES

Geographical origin, distribution & crop losses:

Root-knot nematodes (RKNs) were first reported in 1855 by Berkeley in UK, who observed them causing damage on cucumbers. The infection of RKN in potato tubers in India was firstly documented in Shimla by Thirumalachar in 1951. Several species of RKNs have been reported from infected potatoes however, only six species considered to be destructive worldwide. *M. incognita* is common in the tropical regions followed by *M. javanica* and *M. arenaria* while *M. hapla*, *M. chitwoodi* and *M. fallax* are found in cooler climates and it causes >12.2% yield loss worldwide. The nematodes can interact with other soil-borne fungus, bacteria, and viruses, causing significant crop loss.



Fig. 101: Symptoms caused by root-knot nematode (*Meloidogyne* spp.) on tubers

Symptoms: Stunting and yellowing of plants with chlorotic leaves, galls in roots, warty “pimple-like” lesions on tubers, brown patches on flesh of the sliced tubers (Fig. 101) and tuber deformation of potato tubers. The most important RKN-*Ralstonia solanacearum* interaction causes “pseudomonas wilt”.

Causal organism(s) & Key characteristics: The most widespread and economically important species in potato are *M. incognita*, *M. javanica*, *M. arenaria*, *M. hapla*, *M. chitwoodi*, and *M. fallax*. The sedentary adult root-knot females are pearly white in colour. *M. incognita*, *M. javanica*, *M. hapla* and *M. enterolobii* females are pear shaped without posterior protuberance but *M. hapla* has rough and irregular appearance. *M. fallax* and *M. chitwoodi* has round body with short neck and slight posterior protuberance. Variations in parineal patterns of *Meloidogyne* species are observed. In *M. incognita*, dorsal arch is high without lateral lines while in *M. javanica* it is usually flattened with distinct lateral lines. In *M. arenaria*, dorsal arch is low and rounded, dorsal striae slightly indented forming a shoulder on arch, lateral lines weakly visible; whereas, in *M. hapla* dorsal arch is usually rounded with low dorsal arch, lateral lines clearly appeared. *M. chitwoodi* and *M. fallax* possess round to oval dorsal arch, lateral lines weakly visible.

Disease cycle and favorable conditions: The J2s emerging from the egg masses begin feeding on the young potato roots. Juveniles in the second stage (J2) moult and go through the J3 and J4 stages before becoming adult females or males. Males are migratory and thread like, while adult females are sedentary, and pear shaped. Males exit the root to find and mate with females. Females lay 200 to 1500 eggs in a gelatinous matrix, which commonly adhere to root galls. The length of the life cycle depends upon temperature, during the summer it complete the life cycle within 25-30 days whereas in the winter, it requires 65-100 days to complete the same. The optimal temperature for development and reproduction ranges from 21-27°C.

Management

- ❖ RKN infestations can be reduced by using quality seed tubers, summer ploughing, field sanitization, and keeping the field free from weeds.
- ❖ At least four-year crop rotation with non-host crops such as maize, wheat, oats, cotton, sorghum, grasses, *Brassica* crops such as cabbage, cauliflower, mustard, Chinese cabbage and growing of antagonistic crop (French marigold) in alternate rows with potato will reduce the RKN populations.

- ❖ Some of the cover crops such as rye, clover, castor, beans, chrysanthemums, and sesame suppress nematodes through chemical properties of residues. Sun hemp acts as a poor host for nematode. Organic soil amendments from *Tagetes* spp., *Ricinus communis*, and *Datura stamonium* increase potato tuber yields due to their nematicidal properties.
- ❖ Nematicides such as oxamyl (vaydate), fluopyram (velum prime), fluensulfone (nimitz), ethoprop (mocap), and ethoprophos (nimayuk) are recommended for management of RKNs.

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Name of disease: PHYTOPLASMA ASSOCIATED DISEASES

SHOOT PROLIFERATION

Symptoms: Little leaf, Profuse shoot proliferation in variety Kufri Khyati and Kufri Surya ([Fig. 102](#))

Distribution: Bhubaneshwar (Odisha)

Causal organism: 16SrVI-D subgroup

Disease cycle and Epidemiology: Carrier as phytoplasma strain to other vegetables



Fig. 102: Symptoms of phytoplasma associated diseases in potato: a: little leaf, b: shoot proliferation; c: small and malformed tubers

Nursery Diseases

Damping off is the most important disease of tomato, chilli, capsicum, brinjal, and cole crops. Although seed rotting, bacterial blights, downy mildew, black rot and nematode infestation are other diseases of nursery crops. Several seed and soil borne fungi can kill before the tender radical and plumule are established in the nursery bed. The pre-emergence damping off and seed rotting are the usual causes of poor seedling stand. This type of damping off causes hidden loss but the post emergence damping off is a more serious problem. Seedlings get infected near the soil line after emergence and colonized by fluffy fungal growth. The tissues become soft, water soaked and weak causing the seedlings fall over and later they die. The entire disease progress is so fast that leaves remain green, but they are unable to stand further (Fig. 103). Stem rot near collar region of seedling is another severe problem observed mainly after 20 days of seed emergence (Fig. 104). Damping off is caused by several species of soil fungi where *Rhizoctonia*, *Pythium*, *Fusarium*, *Phomopsis*, *Sclerotium*, are more or less associated with vegetable crops. However, *Rhizoctonia solani* is most severe among all the pathogens. Bacterial leaf spot is second most important seedling disease of tomato and chili. Symptoms appear as very minute, dark spots on all above ground parts. Leaves turn yellow and blighted leaving only stem (Fig. 105). Heavy soil, excessive rain, high soil moisture, dense planting, low light intensity and high dose of nitrogen favour seedling diseases.

Management

Management of damping off disease is only possible by integration of physical, biological, cultural and chemical methods.

Soil solarization

Soil disinfection using white transparent polythene in hot summer is one of the most effective approaches for management of soil borne diseases particularly in nursery beds of high value horticultural crops and greenhouse crops. The main objective of soil solarization is to eliminate pathogens, weeds, toxic metabolites, biotic agents, insect etc. (Fig. 106). Dominant soil pathogens like *Macrophomina*



Fig. 103: Damping off



Fig. 104: Collar rot



Fig. 105: Bacterial leaf spot

phaseolina, *Fusarium oxysporum* f.sp. *pisi*, *Meloidogyne* spp., *Pseudomonas* spp., *Verticillium dahliae*, *Phytophthora* and *Pythium* spp. are effectively reduced in nursery beds. Excellent control of bacterial blight to the tune of 55-60% of seedlings was recorded in solarized beds. It also reduced the population of viable sclerotia of *S. rolfsii* and *Sclerotinia* spp. Soil solarization increases yield in corky root disease of tomato and Verticillium wilt of brinjal. Availability of soil nutrients particularly phosphorus and potash are increased. Organic matter and cruciferous plants addition in nursery beds before polythene mulching have been found more effective in reducing the cabbage yellows, *Pythium* spp and *S. rolfsii*. Effect of solarization further increased when it was integrated with some selected pesticides and neem cake. For effective results, soil solarization performed on a tightly laid white plastic as close as possible on a smooth soil surface. The edges must be tightly held by burying them in soil. Saturate the soil to at least 70 per cent of field capacity before or after laying plastic. The plastic should be left for 4-6 weeks to allow soil to heat to deeper depth.



Fig. 106: Soil solarization with white polythene

Biological control

Biological seed treatment can be done by seed priming, seed coating, seedling dipping, dry powder treatments etc. depending upon the nature of biocontrol agents (Fig. 107). Generally, 6-10 gram *Trichoderma* formulation for one kg of seed is recommended for seed treatments but spore concentration of suspension should be 10^6 to 10^9 /ml. Similarly, 10-25 g powder should be applied in per m² area depending upon of the soil type and organic matter. Seed treatment by *Trichoderma* @1% along with soil application @10 g/m² area is equally good for soil application of *Trichoderma* @10 grams along with 100 grams neem cake/m² for nursery diseases and nematode problems. Some *Trichoderma* spp. is compatible to fungicides at lower doses of pencycuron, copper hydroxide, captan that may be integrated.



Fig. 107: Efficacy of biocontrol agents against soil borne pathogens

Chemical control

The easiest and most effective control measure for vegetable damping off is to drench the nursery soil with captan @0.25% solution. Five-liter solution of this chemical should be uniformly applied in the 1x1 meter² area of nursery bed. After complete germination of all the seeds, copper oxychloride @0.25% should be sprayed thoroughly in such a way that seedling and its root zone must be covered by the solution.

Cultural method

Nursery bed should be properly drained and pulverized. Soil texture of nursery bed should be light and porous by adding equal proportion of sand and compost. Sowing of seeds should be thin enough by maintaining equilateral distance so that seedling will not be crowded. Proper aeration and light to check damping off infection should be provided. Protect germinating seeds and juvenile seedlings from heavy rain and scorching sunlight through agronet, muslin cloth and portable low tunnel polythene arch.

Note